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THE HEART AND ARTERIES IN MID-LIFE*

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The problem of heart disease in and beyond mid-life carries with it many interesting thoughts as one reviews the literature. Since heart disease in these individuals may manifest itself in different clinical entities which have typical syndromes, it is important that we discuss some manifestations of each. Thus, conditions known as primary cardiac failure may occur, but they are usually associated with arteriosclerosis with or without increase in blood pressure. Primary deficiencies of cardiac nutrition, such as lack of essential food substance or endocrine deficiency may be found in this group. Many of these hearts show only diseases of the arteries at autopsy. Yet study of people with heart failure shows, for one thing, a persistent negative nitrogen balance and protein deficiency.

Coronary thrombosis is perhaps the most dramatic and common heart condition found in this age group. The work of Herrick (1) some forty years ago, pointing out the true nature of myocardial infarction as being due to thrombosis of the coronary artery, was heralded as a great discovery in the advancement of medicine. True, it was important, but what seemed to be such a significant discovery at that time has not lead to a simple interpretation. The work of Selye (2), too, can be heralded as an important contribution to the over-all story of heart disease, particularly with regard to emotional stresses and their relationship to acute heart disease. These studies emphasize the step-like advance of human knowledge and control of disease, and also the importance of the fundamental understanding not only of anatomic changes but also of factors which may lead to disease processes in the body. As far as myocardial disease is concerned, particularly myocardial infarction, clinical histories have pointed out that there may be several different factors which produce changes.

A story (3) noted by one of us was that of a 30-year-old ball player who received a direct blow over the heart. He fainted, and died after a few days in the hospital. The autopsy revealed a myocardial infarction with minimal sclerosis of the vessels and a fresh thrombus, suggesting that injury to the heart made through the chest was the important factor in the production of coronary thrombosis. Most individuals who have coronary thrombosis or heart disease past mid-

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life have no such traumatic etiologic factor. It is true that the condition may come on suddenly associated with work or emotional strain without trauma. In some instances these individuals may be in good health and may have had no clinical manifestations of heart disease previously. Some individuals at mid-life and beyond may or may not have clinical symptoms and when death occurs from some other cause, postmortem examination may show complete or nearly complete obstruction of the coronary system without myocardial infarctions.

This discussion serves to indicate that diseases of the heart and vascular system past mid-life may not have a single etiologic factor, even though they may be associated with obliteration of the coronary artery system of the heart. Heart disease in and beyond mid-life is associated with important fundamental alterations in the body. During this age period conditions occur that cause development of various changes which are reflected in the vascular system. This state accounts for the increase in the incidence of heart disease in this age-group, an increase which is manifest more at present than at any other period of time. A prime reason is that more people now live longer, due to the practice of better medicine at an earlier age, and grow to an age when degeneration takes its toll of health. The discussion of the causes of degeneration associated with heart disease would necessitate a detailed review of the causes of arterial and degenerative vascular disease. It is not necessary for us to go into such a detailed discourse of the various theories that have been propounded.

Perhaps we should say that the most logical, likely and satisfactory work in this field is the work of Winternitz (4), who has shown that the nutritional channels of the larger arteries may be the site of morbid changes which cause anoxia and reduced nutrition to the arteries, thereby causing scars and deposits of cholesterol. Hypercholesterolemia alone does not seem to be a primary factor in most individuals with coronary disease. However, probably some other associated factor, such as anoxemia which sensitizes the blood vessels, may be associated with cholesterol deposits. With the realization that fundamental body changes must play an important role in the cause of degenerative vascular disease, it is of necessity that we review some of the more recent work which indicates the probable relationship. It would seem from observations that have been made to date that body nutrition plays an important role in the occurrence of such phenomena.

Murata and Kataoka (5) were the first to demonstrate the important point that thyroid substance fed along with cholesterol in oil prevents deposits of cholesterol in the arterial walls of rabbits. Since their work there has been a gradual increase in observations which implicate thyroid function as playing an important part in vascular degeneration. Goldberg's work (6) in 1927 was conclusive; he removed the thyroid from one of twin lambs and found definite evidence of arteriosclerosis in the experimental animal but not in the other. Little attention was paid to these observations clinically, however, until Bruger and Rosenkrantz (7) in 1942 reported the association of low basal metabolism with increased incidence of arteriosclerosis. The work of Kountz and Hemplemann (8) on individuals who had their thyroids extirpated for clinical reasons indicated degeneration of the arteries and especially of the aorta, a finding which was substantiated by

Foster and Barr (9) in a case of spontaneous hypothyroidism. Recent work in our laboratory (10) has shown that in a series of cases in which the incidence of advanced arteriosclerosis was correlated with the rate of oxygen consumption and age, a decrease in the basal metabolic rate was found to be a more important factor than age itself. Observations have been made by Dungal (11) in which it was found that the incidence of arteriosclerosis in Greenland was relatively nil when compared with its incidence in the same type of individuals in Austria. The intake of iodine and the use of fish protein were thought to be factors.

The work of Katz (12), Barr (13), and Gofman (14), as well as that of ourselves and others, has shown that in conditions of myocardial infarction and coronary artery sclerosis there are changes in the blood, due partly to lipoprotein disturbances. Apparently a high lipoprotein factor in the area of the beta globulin is involved. Gofman has found it by his technic in the S_f 3–8, and 12–20 areas. Perhaps there are other changes of which too little is known at the present time. This work suggests that a biochemical disturbance in the field of the lipoproteins may cause, or at least be associated with, coronary artery degeneration and heart disease in mid-life. Other conditions may also play an important role in vascular and heart disease. The influence of high blood pressure as a cause of arteriosclerosis and coronary artery disease has been pointed out by Farber (15). He suggests that injury with macroscopic and microscopic thickening of the vessel wall may occur in this condition.

Since the lipoprotein factor appears to bear an important relationship to arteriosclerosis and this, in turn, suggests that nutritional changes play a leading part in the cause of the disease, proper control must lie in adequate knowledge of protein and lipid nutrition of individuals. Recent work has shown that many older people, especially those with arteriosclerosis, have a negative nitrogen balance, unless under special care. It is found necessary to increase the protein intake in some individuals, in order to prevent nitrogen loss. Such fundamental disturbances of protein metabolism may be a factor associated with the lipoprotein change found in persons with arteriosclerosis. The work of Lansing and associates (16) indicates that changes in the protein content of the blood vessel walls are common in degenerative vascular disease.

Studies in older individuals reveal that a reduced protein intake may be a factor in the disturbance of protein balance. Diet, however, is not the only concern; steroid hormones, particularly androgens (such as testosterone), insulin and thyroid, are known to assist in retention. Cortisone and ACTH, on the other hand, increase nitrogen loss when administered up to the point at which the appetite is stimulated. After that, there is compensation for the nitrogen loss by increased food intake. According to Katz and associates (17), experimental arterial sclerosis is increased by administration of cortisone and inhibited by administration of androgens. These findings suggest that hormonal balance may have an important influence on the protein and lipoprotein factors of the body which, in turn, influence arteriosclerosis. It can thus be said that recent work in the field indicates that disturbance of nutrition plays an important role in the appearance of arterial degeneration and heart disease. This would apply, therefore, to coro-

nary sclerosis and to associated cardiac failure. It has been shown that lack of endocrine substances, such as thyroid, may lead to myxedema and heart disease; lack of this substance may also lead to arteriosclerosis and certain forms of anoxia of the vascular system. It is believed by some that diabetes may lead to arteriosclerosis and hence to heart disease. Whether or not the lack of sex hormones in the body and their influence on nutrition play an important role in this respect is not definitely known, but it is certainly suggested by recent work.

That sex hormones may modify the blood vessel wall in the uterus of older women has been established in studies in the St. Louis City Infirmary Hospital. Changes in the uterine blood vessel walls were noted and interpreted as showing a lessening of the processes of arteriosclerosis, in tissue obtained post mortem. In the series, both nontreated and treated individuals were examined. A marked degree of arteriosclerosis was always found in the uteri of nontreated, senile subjects. The vessels showed small lumens, thickened walls, and often deposits of calcium. Since these studies were always postmortem examinations, it was necessary to compare the uteri of different women of similar age and physical condition before death, rather than the uterus of one specific woman before and after treatment (Figs. 1 and 2).

This opens up a definitely different approach for the clinical control of heart disease. It is essential that careful clinical evaluation be made of individuals who have heart disease and of individuals at and beyond mid-life who are in the period of body degeneration. Careful study of the body physiology and chemistry is im-

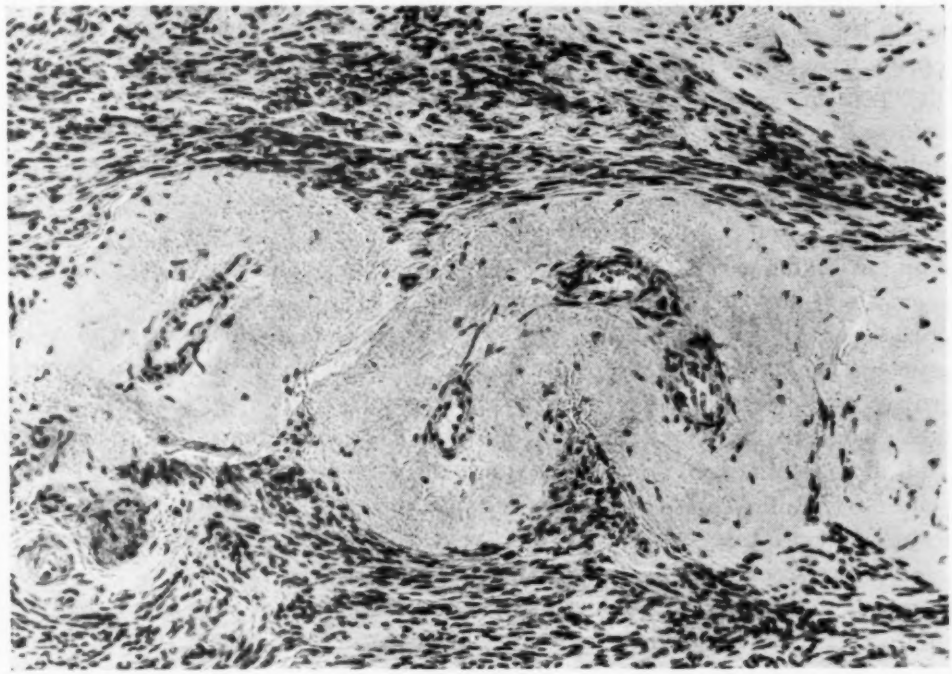

FIG. 1. Photomicrograph of a section of the uterus of a 62-year-old female. Note the group of arteries and the relatively small size of their lumens. Their walls are thick and composed of hyalin, with very few cells. Note the abundance of connective tissue cells.

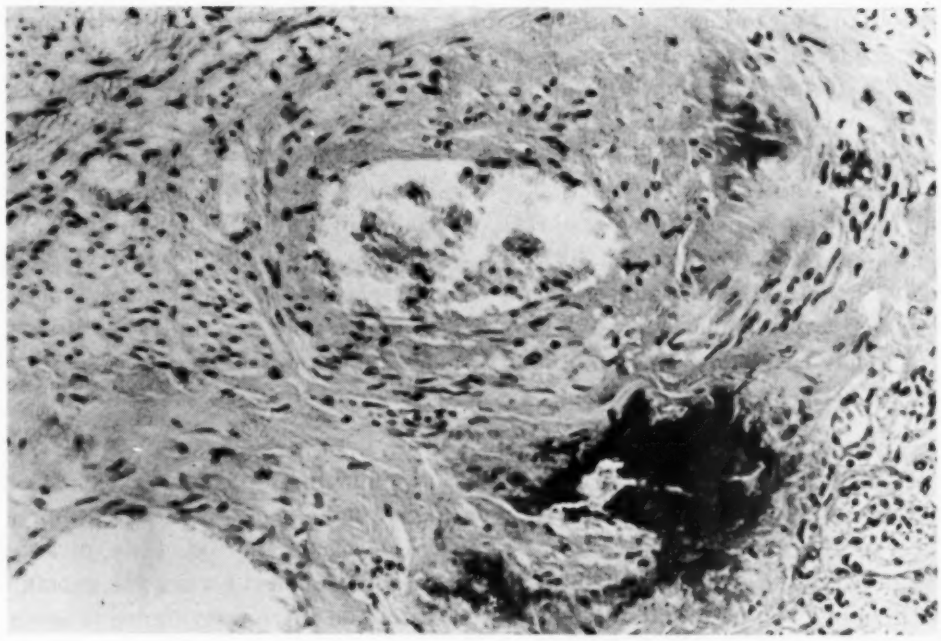

FIG. 2. Photomicrograph of a section through the wall of the uterus of an 82-year-old female who had menstruated monthly for three years. She had received treatment for 38 months. Note the large size of the blood vessels, the reduction in density of the hyalin material and the abundance of living cells in their walls, compared to Figure 1. There is also a relative decrease of the connective tissue. The dark spots are calcium deposits.

portant, in order to maintain a cardiovascular system that functions well and in order to prevent degenerative changes associated with the poor nutrition which is the factor that leads to anatomic disease.

To be more practical, we must ask ourselves what should be done towards the control of degeneration of the body which leads to disease of the vascular system and heart. First, for the ages between 40 and 60 years we can advise yearly examinations, with care taken to evaluate the cardiovascular system and to investigate any major or minor abnormalities. Since in hypothyroidism anoxia plays a role in the development of coronary disease, we may prevent changes by controlling the rate of oxygen consumption as revealed by the basal metabolic rate. This may be accomplished in part by the use of desiccated thyroid, iodine and at least the B group of vitamins, and by the limitation of tobacco, food and alcohol.

We cannot control the faulty lipoprotein metabolism by the administration of protein alone. Sex hormones, androgen and estrogen (need for the latter may be determined by the staining reaction of the epithelium of the vagina or tongue) are important. It is probable that the administration of these substances at a relatively early period in mid-life will not only protect against a lipoprotein disturbance, but will also lessen the load of the adrenal cortex. We may also control nitrogen loss and conserve body energy by giving androgen in physiologic amounts if analysis indicates that the individual is losing nitrogen readily. A diet may be prescribed which is relatively high in protein, moderate in fat and

carbohydrate, and particularly low in sugar if the glucose tolerance curve is persistently at variance with normal. If the blood cholesterol level is above 300 mg. per 100 ml., we should insist that the individual lower his fat intake if it is high; lipotropic substances may be administered.

In the later stage of degeneration between the ages of 65 and 80, we should carry the studies further, making general function tests, and determining the rate of oxygen consumption, the rate of iodine uptake by the thyroid gland, the excretion of 17-ketosteroids (an index of adrenocortical function). Defects in many of these systems may be found and corrected. We can establish the variance of the lipotropic factor from its usual picture and attempt to adjust it by such means as diet and the administration of hormones.

SUMMARY

It should be assumed that individuals past mid-life have certain deficiencies, and that the recognition and correction of such deficiencies is highly important. These changes should be recognized at a relatively early period of life, just as the pediatrician recognizes the changes of growth and the need for growth substances and food as well as for immunization, in childhood. Strong evidence is accumulating that in persons past mid-life nutritional factors of the body are the primary concern of health, and that we may lift the veil from chronic vascular disease as it is known today by paying attention to such matters as the rate of oxygen consumption, endocrine balance and diet in people over 40 years of age.

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THE RELATIVE MERITS OF SERUM LIPOPROTEIN AND CHOLESTEROL DETERMINATIONS IN ATHEROSCLEROSIS

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Within the past few years there has been a marked intensification of investigations in fat metabolism and its disorders. There is growing awareness that atherosclerosis is a metabolic disease in which plasma lipids penetrate into the arterial wall, that it is not an inevitable consequence of aging, and that it may be preventable and perhaps even partially reversible. The ultracentrifugal method of analysis of serum lipoproteins (1) provided much of the stimulus for the newer work in this field. More recently, however, certain investigators (2, 3, 4) have stated that measurements of the blood cholesterol concentration give as much or as little information as determinations of specific lipoproteins (Sf 12–20 and Sf 20–100 classes), and have challenged the statistical evidence (5) which clearly implies that only a particular range of lipoprotein molecules is etiologically involved in atherosclerosis. Indeed, all lipoproteins contain cholesterol and other lipids, and so it has been difficult to understand and accept the thesis that certain specific classes are atherogenic and others not. This question is an important one, since it appears that normally there is a stepwise breakdown of lipoproteins in serum from larger to smaller molecules, a process in which heparin is involved (6, 7). Metabolic blocks may interfere with this conversion at any stage and so lead to an abnormal accumulation of lipoproteins of various sizes. Thus, determination of the role of such factors as diet, hormones or heparin in normal serum lipid metabolism and its abnormalities, in large measure depends upon the differential significance which should or should not be attached to various molecular classes of lipoproteins. A large body of facts exists, apart from clinical statistical correlations, which bears upon this important subject. These facts are widely scattered throughout the literature, however, and so not easily available. For this reason, and since developments in our knowledge of atherosclerosis are significant not only for investigators in the field but for the entire profession, an evaluation of the available material seems worthwhile.

We may first consider the evidence in humans. It has long been appreciated that measurement of the total blood cholesterol content as an index of atherosclerosis is often misleading. Coronary thrombosis frequently occurs in the presence of a "normal" blood cholesterol level. Furthermore the marked sex difference in atherosclerotic disease is not paralleled by any sexual distinction in serum cholesterol levels. Recently Jackson and Wilkinson (8) stated that the total cholesterol/phospholipid ratio is no more useful as an index of atherosclerosis than the discredited total cholesterol itself. With regard to the ultracentrifugal analysis of serum lipoproteins, however, it appears that several salient facts which add

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weight to the significance of this methodology have been widely overlooked. Of first importance is the fact that this method measures molecules containing fat and cholesterol as they actually exist in serum. Cholesterol does not exist as such in the blood, but is present in combination with phospholipid, neutral fat and protein in giant molecules called lipoproteins. These molecules have been seen with the aid of the electron microscope (9). The ultracentrifugal method thus qualitatively separates and quantitatively determines the various *in vivo* species of fat-containing molecules, whereas total cholesterol is a value determined after the biologically existing lipoproteins have been disrupted by the chemical analysis. It is also of interest that the particular classes of lipoproteins which have been related to the genesis of atherosclerosis occur only in very low concentration in children, in young females, and in animal species in whom atherosclerosis does not spontaneously occur (5). In rabbits, for example, they do not appear until a high-fat, high-cholesterol diet has been given for several weeks and atherosclerotic deposits make their appearance. Thus large concentrations of these specific sized particles, the Sf 12–100 classes, are apparently abnormal. Also significant is the fact that there is a definite difference between males and females: prior to the menopause women have considerably less Sf 12–100 lipoproteins than men in the same age group, thus paralleling the distinction in the incidence of atherosclerotic disease between the two sexes. Recently Biggs and Colman (10), using tritium-labelled cholesterol in humans, measured the different cholesterol specific-activity absorption curves following ingestion. They found that these curves were correlated with the serum lipoprotein distribution but not with the serum total cholesterol values. Thus ultracentrifugal analysis of the serum lipoproteins measures a defect in lipid metabolism which can be independent of the total serum cholesterol present. Finally, there is a good correlation between Sf 12–100 lipoproteins and proven coronary atherosclerotic disease, whereas the other lipoprotein classes do not show this relationship.

Animal experiments shed further light on the subject. Several investigators have shown that the degree of atherosclerosis is often poorly correlated with the blood cholesterol level. Eberhard (11) fed rabbits a high-cholesterol diet, with and without the addition of alcohol, and found that despite a more marked hypercholesterolemia in the cholesterol-plus-alcohol group there was a decreased deposition of cholesterol in the liver and aorta. Brown *et al.* (12), in many of their experimental groups, found markedly elevated serum cholesterol levels and no atherosclerosis. Ladd, Kellner and Correll (13) used Tween-80 in rabbits and produced considerably higher cholesterol levels than in controls, but less atherosclerosis occurred. Duff and Payne (14) and McGill, Parrish and Holman (15) demonstrated that alloxan diabetic rabbits fed a high-cholesterol diet show a markedly elevated concentration of serum cholesterol, but no atherosclerosis is produced. Stamler, Pick and Katz (16) using estrogens plus androgens, and estrogens alone, found that coronary atherosclerosis was markedly decreased in cholesterol-fed cockerels, although there was no change in the degree of hypercholesterolemia. Apparently in experimental animals, as in man, an elevated serum cholesterol level does not necessarily indicate atherosclerosis.

However, there is evidence that lipoproteins of a certain particle size are well correlated with atherosclerosis. Bragdon (17) injected hyperlipemic rabbit serum into other rabbits and atherosclerosis was found in the recipient animals within forty-eight hours. When the top fraction of the lipemic serum (containing the largest sized lipoproteins) was used, no atherosclerosis was produced. The lower lipemic fraction (containing smaller lipoprotein classes) was atherogenic. Apparently, since both top and bottom fractions contained cholesterol and other lipids, specific molecular size was an important factor in atherogenesis. Pierce (18), using alloxan diabetic cholesterol-fed rabbits, clearly demonstrated that particle size is of great importance. These rabbits all showed markedly elevated serum cholesterol levels but many had only minimal atherosclerosis. There was a negative correlation between atherosclerosis and serum cholesterol concentration, but a significant positive correlation between lipoproteins of the Sf 12–30 class and atherosclerosis. There was a significant negative correlation between atherosclerosis and lipoproteins of Sf greater than 100. Since a high proportion of the serum cholesterol in the alloxan diabetic cholesterol-fed rabbit is transported in lipoprotein classes above Sf 100, the lack of positive correlation between serum cholesterol concentration and atherosclerosis is easily understood. Evans, after numerous studies using his aortic perfusion technique (19), stated that only lipid of a specific particle size is deposited in aortic strips.

Woerner (20), using injections of various lipids in guinea pigs, also showed that the range of particle size is of great importance in the deposition of lipids. No fatty acids of less than 20 carbons were deposited; above this size, deposition occurred. When the droplet size was increased above the range in which deposition occurred, the amount of lipid deposited was not increased.

Hueper (21), using acacia, pectin, methyl cellulose and polyvinyl alcohol, clearly demonstrated that certain macromolecular substances damage vascular walls in a manner closely resembling arteriosclerosis. He believes that these agents are harmful, since macromolecules do not pass readily through the normal filtration membranes, *i.e.*, the capillary walls, and are therefore not rapidly eliminated through the usual channels. The size of the pores in vascular walls and in other filtration membranes controls the movements of macromolecular substances in the living organism. He also believes that the molecular shape, apart from the molecular size, exerts a similar influence and determines to some extent the physicochemical relations which may develop and the reactions which may ensue between a particular macromolecular compound and its biologic environment. His observations indicate that atherosclerotic changes are attributable at least in part to a disturbance of the macromolecular equilibrium of the plasma.

Experiments using pectin have been particularly clarifying. Bryant, Palmer and Joseph (22) demonstrated that when pectin was autoclaved, its molecular weight was reduced to the molecular weight of plasma proteins. This autoclaved pectin of lower molecular weight did not accumulate in body tissues, whereas unautoclaved pectin sols, containing much larger pectin molecules of the same chemical composition as the smaller ones, were retained in body tissues. Hueper (23) found in rabbits and dogs that intravenous freshly prepared pectin produced

marked foam-cell atheromatosis, whereas heated pectin caused only very minor hyaline arterial lesions. He concluded, "these findings indicate that the marked reduction in the molecular weight and size of the pectin molecules produced by exposure to prolonged heat resulted in increased and relatively rapid excretion of the injected macromolecular material or in progressive metabolic degradation to still smaller molecular units easily penetrating the filtration membranes. It is thus demonstrated that the production of atheromatosis and thesaurosis by the injection of pathogenic macromolecular substances depends on the size of the molecules and on the metabolic stability of these substances."

Pappenheimer, Renkin and Borrero (24), in studying capillary diffusion, found that capillaries in different regions of the body vary greatly as to their permeability characteristics. In cats the hindlimb capillaries have an average pore radius of 30 Angstroms, whereas glomerular capillaries have a pore radius of perhaps 50 Angstroms. They showed that restricted diffusion is a function of molecular size, that molecular sieving occurs, and that with increasing molecular size there is decreasing diffusion.

There is good evidence that vascular walls act as selective molecular filters, as clearly demonstrated by Wilens and McClusky (25). They perfused segments of excised arteries and veins with blood serum and determined the blood proteins and the total and free cholesterol of the original sera, the filtrates, and the fluids remaining in the vessel lumen. It was found that both arteries and veins are almost freely permeable to small molecular substances such as India ink and egg-yolk cholesterol. There is, however, another group of substances of intermediate molecular size, only fractions of which filter through. In this group, which includes cholesterol, there is an inverse relationship between molecular size and filterability. It was noted that the combined amount of protein or cholesterol in the filtered and nonfiltered serum fractions after transarterial filtration was less than would be expected in an equivalent volume of the original serum. Thus some of these substances must have been retained in the arterial wall. This inference was substantiated by lipid stains of sections of the artery which clearly showed intimal and medial lipid. Dye studies with Evans blue and Trypan blue also demonstrated deposition of these materials in the arterial wall. Veins were much more permeable to serum proteins and lipoproteins than arteries, thus suggesting that this difference in structure and permeability accounts for the difference in incidence of spontaneous lipid deposition. Evans (26), using carbon particles of varying size, also found that the smallest units passed completely through the arterial wall, that particles of intermediate size penetrated into the arterial wall and were trapped intramurally, and that the largest sized particles were unable to enter the intima at all.

DISCUSSION

Ultracentrifugal analysis of blood serum has shown that serum lipids are carried in large molecules of different sizes, which contain varying amounts of free and ester cholesterol, phospholipid, neutral fat and protein. Studies of different macromolecular substances indicate clearly that particle size is a factor of great

importance in the pathogenesis of vascular disease. Experiments in hypercholesteremic and alloxan diabetic rabbits demonstrated that only a portion of the lipoproteins, not the total cholesterol, shows good correlation with atherosclerosis. Techniques using arterial filtration by blood serum provide evidence that the vascular wall is semipermeable, and that molecular size in relation to porosity is of first importance in determining the passage through, or the intramural entrapment of substances. A portion of the serum cholesterol (about 20 per cent) filters through excised arterial wall, part is deposited in the arterial wall, and the rest remains in the vascular lumen since it cannot penetrate into the arterial wall (25). It is the smallest lipoproteins which pass through the vascular wall into the lymph. The largest lipoprotein particles cannot enter the arterial wall at all, and the groups of intermediate size are caught intramurally. Although the definitive experiment, which is to study the take-up by artery segments of perfused individual lipoprotein classes, remains to be carried out, it is highly probable that the lipoproteins which Gofman *et al.* (5) consider atherogenic fall into the intermediate group. All the available data, taken together, suggest that this is true. The evidence is also strong that different portions of the vascular tree vary in their permeability characteristics. It seems probable that, given equal pressures, variations in permeability and the amount of different lipoprotein classes in the circulating blood largely account for the extent and distribution of atherosclerosis throughout the body. Enzymatic removal mechanisms for entrapped lipid (27) undoubtedly play an important role, but we know very little of these factors. At any rate, since probably only that portion of the blood cholesterol carried in specific particle sizes becomes deposited in arterial walls, it follows that the ultracentrifugal measurement of these particular lipoproteins affords more specific information regarding atherogenesis than determinations of the serum cholesterol. This is especially true since Sf 12–100 lipoproteins and cholesterol do not parallel each other in a fairly large percentage of patients (perhaps 25 per cent).

SUMMARY

This paper reviews the evidence currently available in the literature bearing upon the relative merits of serum lipoprotein and cholesterol determinations as indices of developing atherosclerosis. It has been amply proved in animals and man that the total cholesterol level is often misleading in this regard. Certain lipoprotein classes are well correlated with the incidence of the disease in man. In several different experiments these lipoproteins were correlated positively with atherosclerosis, whereas cholesterol was not.

Earlier work with various substances showed that a particular particle size is associated with the development of atherosclerosis. Studies of capillary permeability indicate that restricted molecular diffusion occurs through capillary walls and that molecular sieving takes place. The vascular wall acts as a selective molecular filter. A logical explanation is thus afforded for the apparently paradoxical fact that only those lipoproteins of a particular size-range are atherogenic. Since the blood levels of total cholesterol and Sf 12–100 lipoproteins (which are probably those particles which by virtue of their particular size become entrapped

in the vascular wall) frequently do not parallel each other, it follows that the determination of Sf 12–100 lipoproteins is better than the determination of cholesterol as an index of atherogenesis.

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EMPHYSEMA IN THE AGED

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INTRODUCTION

The clinical and anatomic condition recognized as emphysema is a common one, and as such presents many problems. Diagnosis of this pulmonary change is usually easy, but the understanding of its development is often difficult. In some instances, however, emphysema may be attributed to a simple or obvious cause and the mechanism by which the pulmonary anomaly occurs may be easily explained.

Twenty years ago emphysema was classified into two main types: obstructive and nonobstructive (often senile) (1). It was reported that "lungs from patients with senile emphysema collapse when the chest is opened, and are not enlarged if the barrel chest is associated with a straight spine." Then it was pointed out that if kyphosis be present "more or less true emphysema results." Furthermore, obstructive emphysema in a young person was usually thought to be due to an allergy. Older patients with chronic lung disease may show either the obstructive or the senile type of emphysema, or a combination of both. Illustrations have been published which emphasize the importance of the skeletal contour. Changes in the shape of the thoracic spine (*e.g.*, dorsal kyphosis) lead inevitably to an enlargement in the space of the intrathoracic cavity, a gradual dilation of the lungs and finally emphysema. However, the implied pulmonary insufficiency is not proportionate to the anatomic change. A pertinent misconception is that there is little loss of respiratory function in senile emphysema. During the past fifteen years new methods of study have been devised, which have made it possible to evaluate more accurately the degree of integrity of pulmonary function (2).

PATHOLOGIC PHYSIOLOGY

In emphysema there is a marked loss of elasticity of the lungs. The resting position of the chest is normally determined by the balance between the elasticity of the lungs, and the elastic properties of the chest wall. Therefore in patients with emphysema, the resting position is displaced towards that which normally is assumed during the inspiratory phase of respiration. A second, and very important defect is an increased resistance to the free flow of air in the lungs. This resistance is most marked during expiration. Because of this defect, and the lost elasticity of the lungs, the emphysematous patients find breathing very difficult. The handicap is enhanced by the fact that the circulation of the blood is greatly diminished in many of the affected alveoli and in most bullae. This leads to an abnormal ventilation-perfusion relationship, not only in the diseased parts of the lung, but also in the functioning lung mass (3). It has been amply demonstrated

that advancing age is accompanied by a decrease in pulmonary function (4) and in the pulmonary capacity. In elderly people there is an increase in the residual air and mid-capacity. If a patient is 50 years old or more, his age must therefore be considered when his pulmonary capacity is determined in absolute values (5).

The impaired alveolar ventilation results in a decrease of the O_2 tension, and in an increase in the CO_2 tension of the arterial blood. These biochemical alterations, particularly of the pH of the blood, do not parallel the degree of emphysema. This is due mainly to the fact that the increasing CO_2 tension provokes a corresponding rise in the bicarbonate content of the blood. However, certain complications, particularly infections, may bring about a sudden rise in the CO_2 tension which leads to severe respiratory acidosis and death.

Patients with emphysema may also die of right sided heart failure. This results from a progressive deterioration of the pulmonary vascular bed, which leads to the syndrome known as cor pulmonale. We had the opportunity during the past twenty-three years to study 31 patients with emphysema in whom cor pulmonale developed; they presumably died in a state of congestive failure. At autopsy all of these patients showed varying degrees of pulmonary fibrosis. The fibrosis was either patchy or continuous, but always present throughout most lung segments. We believe, therefore, that emphysematous patients with cor pulmonale also suffer from pulmonary fibrosis, even though the latter may not be recognized by x-ray studies. Elderly patients with emphysema are in the age group in which other significant forms of heart disease occur, either hypertensive or arteriosclerotic. It has been stated by some clinicians that such diseases of the circulation are more likely than cor pulmonale to be the cause of heart failure in patients with emphysema (6).

MATERIAL STUDIED

This report is based on the observation of 24 patients. These people were chosen because it was feasible to subject them to adequate investigation. Since we are mainly concerned with the problem of emphysema, insignificant details of the histories and of the physical and laboratory findings are omitted. The patients reported in detail are those who illustrate any important clinical and therapeutic problem.

Our group comprised 3 females; the youngest was 34 and the oldest 63 years of age. There were 26 males; the youngest was 35 and the oldest 71 years of age. Of this group, 5 were 35 to 50 years of age, 10 were 50 to 65 years of age, 5 were over 60 years of age, and 6 were older.

The ratio of males to females was 21:3, and is perhaps important. It is another example of the greater male morbidity in our way of life. Another cause may be the greater necessity of the male to remain physically active. The man in this age group, whose principal complaint is breathlessness on exertion, seeks medical advice more often than a woman of comparable age.

All of these patients were studied thoroughly in the hope of finding a means to control their main concern, emphysema.

METHODS OF STUDY

There are many ways to investigate emphysema: 1) physical, and 2) biochemical. Both of these methods, as well as others, entail a physiologic approach. The results obtained must be evaluated in the light of what is known of the normal behavior of the lung mass (tissue and accessories) (7) and of the changes in the function of the diseased lung.

1. The physical method

The examination of the patient usually yields very important information. The patient with emphysema dislikes exertion, and is often unable even to walk. There is no breathlessness at rest. Cyanosis may be present, and this coloration may be modified by a plethora, a hyperemia of the mucous membranes (particularly of the bulbar conjunctivae) and a dilatation of the superficial vessels of the face. The chest is often rounded. This results in an increase in the A-P diameter; the sternum is usually more elevated than normal (1 to 3 inches), and the dorsal spine may be kyphotic. If kyphosis is present, the chest space is markedly increased. On auscultation of the chest the breath sounds are very distant. These patients often have a purely segmental type of emphysema; as they grow older, the lung changes usually lead to those seen in a generalized form of pulmonary dilatation (Fig. 1). Other patients retain a straight dorsal spine but the sternum becomes elevated. This sternal elevation increases the intrathoracic space, which must be filled. It becomes occupied by the most resilient available tissue, which is that of the lungs. The position of the diaphragms varies in these people but the motion of the diaphragms is usually good. Roentgenograms and fluoroscopic examination are not helpful or diagnostic as a rule. However, valuable information can be obtained by roentgenograms taken during a full inspiration when compared to those taken after a complete expiration. This procedure will often show the degree of air exchange which is possible in the affected lungs ("clouding") particularly in the lower lung fields (Figs. 2 and 3). In older people there is another form of emphysema which is not uncommon. It is the bullous type; it may be localized, involving only a portion of one lobe of the lung, or it may be present in all of the subdivisions of the lung. The pulmonary insufficiency will often be dependent upon the degree of the anatomic change. In some instances a complication such as infection will make a person with a localized pulmonary bullous emphysema, more of a cripple than the patient who has many scattered bullae which have not been infected. The physical examination of patients with bullous emphysema will disclose at times the obvious shortness of breath on exertion, and at rest. However, some patients with this type of emphysema will show surprising lack of respiratory embarrassment if the bullous anomaly is massive and unilateral; and if it is delineated by a well organized interlobar fissure, the physical signs suggest a pneumothorax. The history and the relatively minor respiratory embarrassment at rest, should help in the differential diagnosis (Figs. 4, 5, and 6). Roentgenologic studies, therefore, assist in evaluating the degree of pulmonary emphysema. Fluoroscopy is essential, particularly in

FIG. 1. Note marked kyphosis of dorsal spine. Initial segmental emphysema developed nto generalized emphysema.

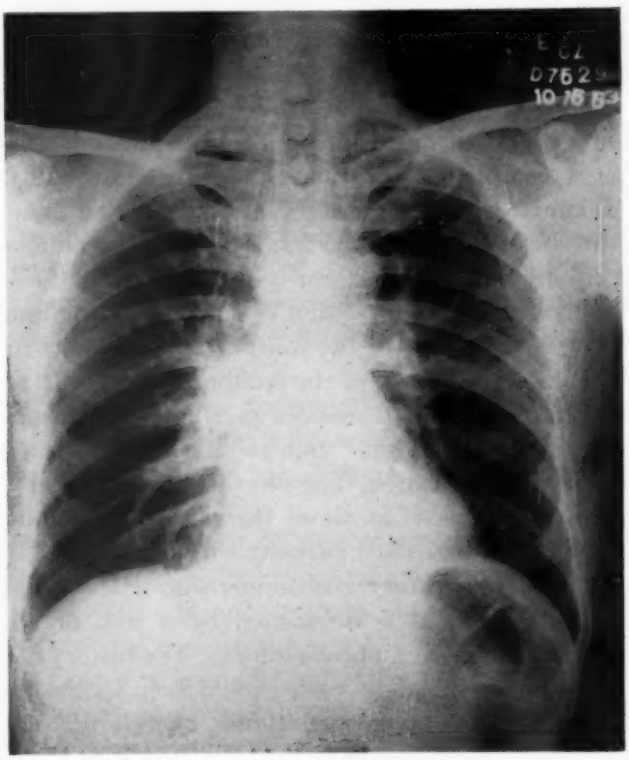

FIG. 2. Note low diaphragms, peribronchial thickening, and horizontal ribs. (Expiration.)

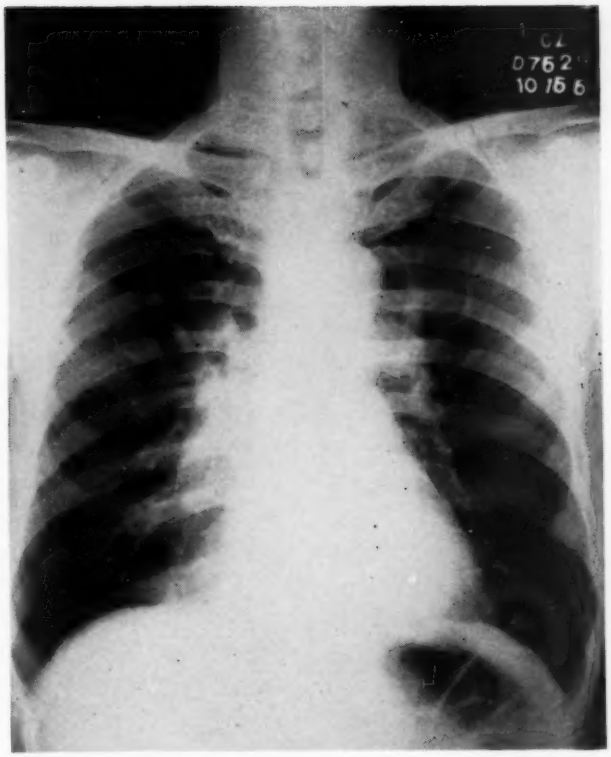

FIG. 3. Same patient as in Figure 2. Note the minimal aeration of the lungs with very little descent of the diaphragms. (Inspiration.)

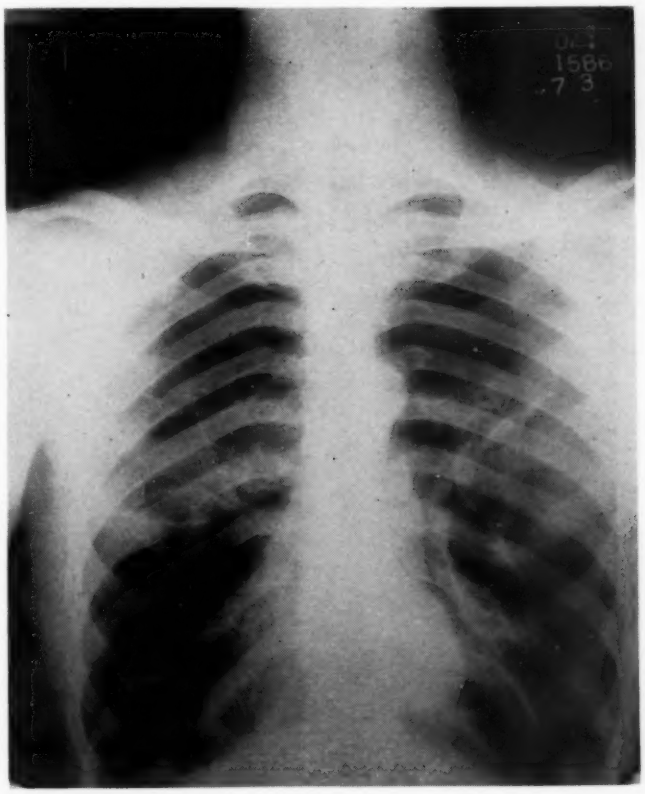

FIG. 4. Note large bullae in the lower portions of the lungs. The parenchymal thickening outlines the bullous dilations.

FIG. 5. Large right sided emphysematous bullae presenting anteriorly

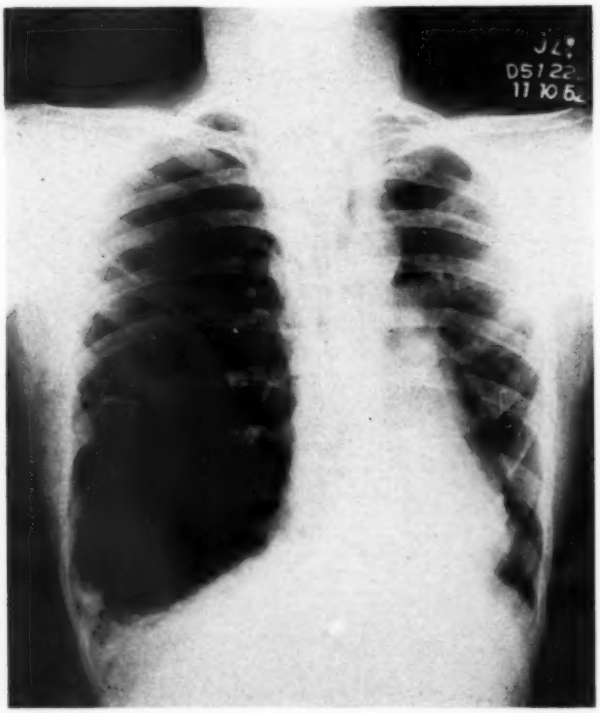

FIG. 6. Same patient as in Figure 5, showing emphysematous bullous degenerations. Heart and mediastinum shifted to left, suggesting at first glance a right sided pneumothorax.

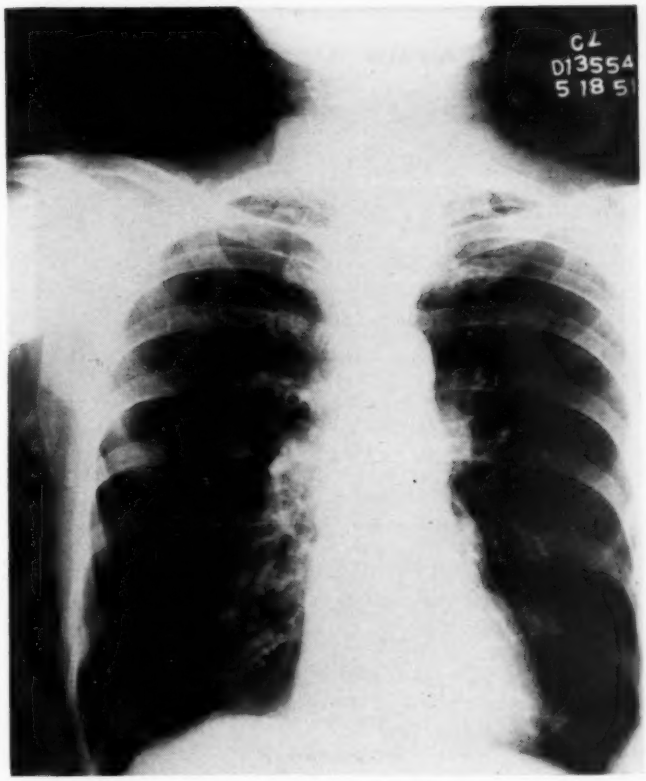

FIG. 7. Patient with emphysema. Note low diaphragm on dark lung fields during full expiration.

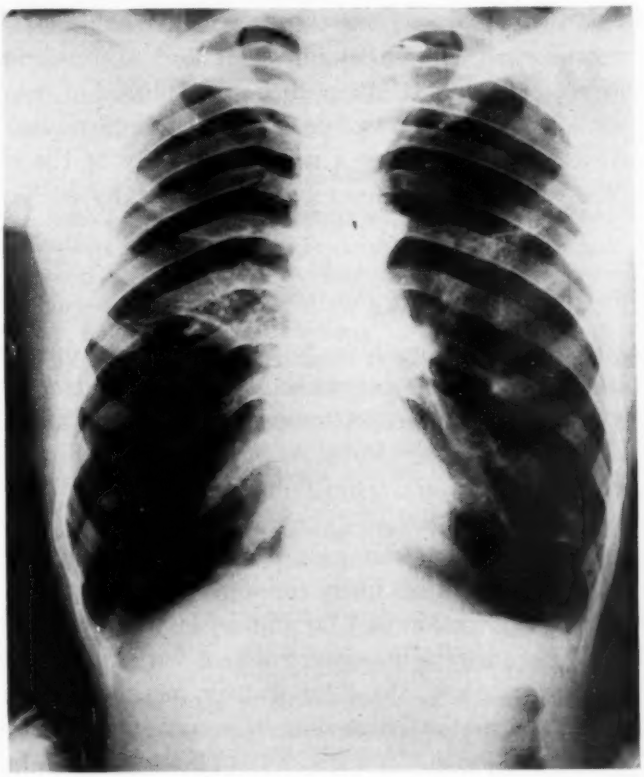

FIG. 8. Routine x-ray film of a patient with senile emphysema, showing peribronchial thickening, and a long straight heart. (At rest.)

FIG. 9. Lateral view showing low diaphragms and peribronchial thickening. (Expiration.)

estimating the movement of the diaphragms and the pulmonary aeration. Similarly, x-ray films of the chest taken during the phases of respiration provide an objective approach in the study of patients with emphysema (Figs. 7, 8 and 9). Emphysema can be detected by a radiologic study of the chest. However, the severity of the emphysema cannot be determined by such physical procedures (8).

2. Biochemical methods (pulmonary function tests)

Purely physical studies are inadequate to determine properly the degree of pulmonary insufficiency in a patient with emphysema. In recent years laboratories have been established in medical centers to study by biochemical methods the most minute functional aberrations. At our Medical School, these investigations were made possible by the Cardio-Pulmonary laboratory directed by Doctors John Hickam and Emil Blair.

Biochemical assay in a person with pulmonary insufficiency entails an analysis of the gaseous exchange which his lungs can accomplish. It is of critical importance to define the concentration of CO_2 and O_2 in the alveolar blood and gas. This is very difficult, because the diffusion gradient varies in the separate portions of the lungs (9). Nevertheless, these laboratory methods make it possible to obtain more direct information regarding the composition of gas and blood entering and leaving the lungs.

TABLE 1

Laboratory Data

	Males	Females
Total no. of patients	20	15
Age range	50–69	
Body surface area (M^2)	1.80	1.70
Vital capacity (VC) (liters)	3.4	2.4
Maximum breathing capacity (MBC) (liters/min.)	91	74
Functional residual capacity (FRC) (% of total capacity)	55	50
Residual capacity (RC) (% of total capacity)	35	35
Total capacity (TC) (liters)	5.6	4.8
* * * *		
Ventilation (L/min./M^2 (rest)	3.9	3.4
Oxygen consumption (cc./min.) (rest)	132	129
Oxygen removal (cc./L of air breathed) (rest)	39	45
Physiologic dead space (cc.)	The physiologic dead space increases with age in the adult	

Table 1 shows some of the many studies carried out in our Cardio-Pulmonary laboratory, and the *average normal* values obtained for people past 50 years of age. These data were supplied by Dr. Blair. The recorded studies were completed on most of our 25 patients. They were repeated whenever possible, on the same patient, in an attempt to evaluate the clinical course and the therapeutic results. It serves no purpose to report the outcome of such investigations in each individual in our group of 25. Therefore, we shall cite only specific examples which illustrate the advantages of pulmonary function tests.

Case Reports

Patient # 1, W. S., was a white male real-estate salesman, aged 58 years (Duke # D-57061). He had progressive exertional dyspnea without orthopnea, of ten years' duration. A paroxysmal cough productive of whitish or yellowish sputum, with severe wheezing, began two years before admission to hospital. Increasing disability led to marked curtailment of physical activities six months after the "asthma" began. The patient was large framed, but not obese, and he had obvious exertional dyspnea with slight orthopnea. There was moderate cyanosis of the lips, ear lobes and fingernail bed. He had a rounded chest with a high sternum and slight kyphosis of the dorsal spine. There was fair equilateral expansion of the thorax, and low diaphragms which moved poorly. The lungs were essentially clear, but the breath sounds were distant. The heart was small, and the B.P. was 140/84. The abdomen was flat and well supported.

Fluoroscopy and x-ray films of the chest confirmed the reported physical findings.

Clinical Impression: Recurrent tracheobronchitis, intrinsic asthma, and emphysema of the mixed type, mainly obstructive.

Dr. Blair's studies are shown in Table 2. His comment and conclusion were: "There is marked reduction in MBC and significant in VC. The patient traps significant amounts of air, in this instance, 539 cc. The AVI suggests an obstructive phenomenon as the underlying cause of the insufficiency. The residual air is significantly increased. All these findings are compatible with a severe degree of emphysema and/or asthma. Conclusion: Pulmonary ventilatory insufficiency, severe."

TABLE 2

Pulmonary Function (Patient # 1)

Ventilation

MBCp—95.8 L/min.*	FRC—4.21 L
MBCo—37.7 L/min. (39.4%)	RC—3.46 L
VCp—3.393 L	TC—5.53 L
VCo—2.073 L (52.7%)	RC/TC—62.5%
IR—1.285 L	ERc—0.207 L
ER—0.207 L	TV—0.582
AVI—0.75	

* MBC—Maximum breathing capacity; VC—Vital capacity; "P"—Predicted value as V.C.p.; "o"—Observed value as V.C.o.; I.R.—Inspiratory reserve; E.R.—Expiratory reserve; E.R.c—Expiratory reserve calculated; T.V.—Tidal volume; A.V.I.—Air velocity index (expression of ratio of the % MBC to the % V.C.); F.R.C.—Functional residual capacity; R.C.—Residual capacity; T.C.—Total capacity; R.C./T.C.—(Residual capacity/Total capacity).

TABLE 3

Pulmonary Function (Patient # 2)

Ventilation

MBCp—123.7 L/min.	FRC—5.20 L
MBCo—63.9 L/min. (51.6%)	RC—3.70 L
VCp—3.96 L	TC—7.60 L
VCo—3.92 L (99.2%)	RC/TC—48.7%
IR—1.618 L	
ER—1.513 L	
TV—0.829 L	
AVI—0.76	

Blood gas (resting)

Oxygen saturation—97.5%
CO_2 tension—47.3 mm. Hg
pH—7.41

Patient # 2, T. J., was a white male, aged 59 years (Duke # D-65168), who had gradually progressive exertional dyspnea over a nine-year period. There were only occasional mild paroxysms of a nonproductive cough, without noticeable bronchospasm. Examination showed a slender, slightly cyanotic man in no distress at rest, but moderately dyspneic on exertion. The chest was rounded, the sternum high. The lateral excursion of the thorax was very limited. The spine and shoulders were straight. The diaphragms were essentially normal in position and moved well. The lungs were clear, but the breath sounds were somewhat distant. The heart was normal, and the B.P. was 150/90. The abdomen was slightly protuberant, but well supported.

Fluoroscopic examination of the chest and x-ray films confirmed the clinical findings.

Clinical Impression: Emphysema, nonobstructive, moderately severe.

The pulmonary function studies made by Dr. Blair are shown in Table 3. His analysis of these data is as follows: "The MBC is considerably reduced, while the VC is within normal limits. This is a common finding in emphysema. The residual air is markedly increased, as is the total lung volume. This again is compatible with pulmonary emphysema. The blood gas

studies are within normal limits." He concluded that this represented an instance of a moderately severe pulmonary ventilatory insufficiency.

TREATMENT

The treatment of elderly patients with emphysema poses many problems. The therapeutic approach is necessarily complex, and cannot be standardized. Moreover, it is important that it be individualized for each patient. The following are some important factors in the treatment of emphysema:

a) *Education of the patient*

The majority of these individuals who seek medical care are males. They have usually led an active life, relying upon their physical efficiency. When the symptoms of emphysema overcome them they are unable to understand their increasing disability, which often condemns them to a sedentary existence. Although many such patients (about 1 out of 3) have known the handicap of recurring attacks of bronchitis and asthma, they find it difficult to accept the continuous respiratory distress caused by emphysema. In our group of 25 patients, asthma had developed in 10 of them before the age of 20 years, and it had occurred off and on since then. Eight patients had begun to suffer from asthma after the age of 40 years, and 7 had never regarded asthma as a primary or important disturbance. It must be emphasized that few, if any patients with emphysema escape a cough which may lead to bronchospasm. This is not influenced by the age at which the symptoms of emphysema develop, nor necessarily by the factors which predispose to, or cause the emphysema. Many such individuals are called "asthmatics", and are treated erroneously as suffering from "allergy." It is essential, therefore, that the physician as well as the patient appreciate the fact that the (asthmatic) bronchospasm is an evanescent symptom, whereas emphysema represents an irreversible alteration of the lung. This can be illustrated by the clinically polymorphic picture known as the "*Postural Syndrome*" (10). Patients who have this postural anomaly are often overweight, and present a large pendulous abdomen. Their complaints and symptoms are many; these are often due to the abnormal spinal curvatures which result in compression of the nerve roots. However, their main concern is increasing breathlessness on exertion. The breathlessness is finally noted even after the little exercise entailed by sitting up, or bending over. They almost never have breathlessness at rest, or orthopnea, and their emphysema is not obvious at first glance. These people are liable to have recurrent upper respiratory infections, and they must be instructed to guard against them. They have frequent paroxysms of coughing, regardless of the presence or absence of infection. These paroxysms usually lead to bronchospasm with audible wheezing, which may mark such people as asthmatics. They do not need an elaborate anti-allergic regimen, since rather simple physiotherapeutic measures, including weight reduction, will rehabilitate most of them.

b) *Physiotherapy and Diet*

These are very important facets of the long-range treatment program of patients with emphysema. The success is dependent on the education of the

patient, and mirrors his personal efforts. General hygienic measures are essential. These comprise the avoidance of unnecessary irritants of the respiratory passages. Smoking is contraindicated. Proper house-heating systems, providing adequate moisture, should be installed if possible. The bedroom of an emphysematous patient, particularly if he is prone to paroxysms of coughing, should have a high moisture content.

If the patient is overweight, he must adhere to a sane and conservative reduction diet. Some of these patients are hypertensive, and perhaps show evidences of renal or cardiac embarrassment. These individuals will profit from a salt-free or a low-sodium diet. It must be kept in mind, however, that many of these patients are elderly, their eating habits are fixed, and they often resent a dietary regimen which is too strict. A considerable number of elderly people with emphysema (8 of our group of 25) lose a great deal of weight and become emaciated. This is usually due to the severe breathlessness which makes it impossible for them to partake of a full meal. Because of this handicap, they finally lose all desire for food. They will benefit from a dietary regimen which divides their food intake into five smaller meals of equal caloric value. The majority of these patients can then be induced to eat again and can partake of 2,800 or 3,400 calories a day.

Patients with a large, pendulous abdomen require a proper abdominal support. Adequate abdominal "belts" are difficult to obtain. They are either too comfortable to be effective, or too cumbersome to be worn indoors and in hot weather. It is obvious that no such support should be fitted until the patient has lost a satisfactory amount of weight.

Breathing exercises provide the most effective physiotherapeutic tool for patients with emphysema. In recent months many articles have appeared in the medical literature regarding these "breathing and postural exercises" (11, 12). During the past ten years at Duke Hospital such physiotherapy has proved to be a very valuable adjunct in the treatment of emphysema. The principle of these exercises is to minimize, inspiration, which is the active respiratory phase, and to emphasize the normally passive expiration of air. These exercises tend to increase chest expansion, vital capacity and maximum breathing capacity, and to decrease the volume of the trapped residual air and the dead space. The results are particularly gratifying in patients who suffer from the nonobstructive type or from the mixed type of emphysema, and in those who present an example of the postural syndrome.

It has been attempted to support the sagging diaphragms of the patient with obstructive emphysema, by injecting oxygen into the peritoneal cavity. The resulting *pneumoperitoneum* is of relatively short duration, and requires a "refill" at weekly intervals during the first two or three months. The procedure is, therefore, rather expensive and impractical if the patients live a long distance from the clinic offering these services. An induced pneumoperitoneum was effected in 4 of our 25 patients. All 4 suffered from severe obstructive emphysema. Two of these men received more than one injection of oxygen—the first two injections were given over a two-week period, and the other four injections were given at approximately ten-day intervals. One of these 2 patients improved dramatically after

the second injection. He came into the dispensary in a wheel chair and insisted on walking out of the clinic after treatment. The following is an outline of his history:

Patient #3, W. D., was a white male, aged 57 years (Duke #D59382). He had been very susceptible to head and chest colds for many years. His familial and past personal histories were negative for the common allergic disorders. At the age of 30 (twenty-seven years previously), he began to suffer from recurrent episodes of non-seasonal asthma. These were initiated as a rule by respiratory infections, which led to paroxysms of coughing. The cough was productive of varying amounts of a tenacious whitish or yellowish material. During the previous three years the exertional dyspnea had led to a marked curtailment of his activities as a dairy farm foreman.

The physical examination revealed a well developed, lean man who was very short of breath, especially on exertion. The chest was rounded, the sternum was high, and the dorsal spine was moderately kyphotic, although the shoulders were straight. The lateral thoracic expansion was practically nil. The diaphragms were very low, and the motion was markedly limited. The breath sounds were distant, and often obscured by late inspiratory musical rales. Sticky inspiratory rales were heard at the lung bases. The heart was normal in contour, although P_2 was accentuated and the neck veins were congested. The B.P. was 152/90. The abdomen was not prominent, but it was relaxed.

Fluoroscopic and x-ray examinations disclosed a marked generalized emphysema, with low fixed diaphragms.

Clinical Impression: Recurrent tracheobronchitis, intrinsic (infectious) asthma, and obstructive emphysema with progressive pulmonary failure.

The patient was studied in the Cardio-Pulmonary laboratory, and the results are shown in Table 4. These results indicate a marked reduction of MBC and a moderate decrease in

TABLE 4

Pulmonary Function (Patient #3)

Ventilation

		After aminophyllin
MBCp—103.6 L/min.	IR—0.435	0.663
o—23.4 L/min. (23%)	ER—1.202	0.891
Aminophyllin, 3 gr., i.v.	TV—0.995	0.871
o—22.1 L/min.		
VCp—3.89 L	AVI—0.35	
o—2.57 L (66.1%)		
Aminophyllin		
o—2.43 L	RC/TC—52.1%	
FRC—3.99 L		
RC—2.79 L		
TC—5.36 L		

Blood gas (resting)

Oxygen content	19.2 vol.%
Oxygen saturation	95.4%
CO_2 (arterial)	50.2 vol.%
CO_2 (serum)	61.2 vol.%
CO_2 tension	44.5 mm. Hg
pH	7.4

VC. The administration of aminophyllin produced very little change in the pulmonary function. However, the patient showed a subjective improvement, but this is not proved objectively by the foregoing data. There is evidence of an increased dead space as shown by the RC/TC ratio. The normal ratio is below 40 per cent. The AVI (air velocity index) is suggestive of an obstructive mechanism as the basis for the pulmonary insufficiency. The results of the blood gas studies were considered to be within normal limits.

This patient had a relapse three weeks after his marked improvement. Despite another treatment he remains, to date, a pulmonary cripple.

The second patient showed no improvement after he had received two injections of oxygen intraperitoneally. He refused further treatment of this kind. The results in the other 2 patients were so indecisive that no conclusions could be drawn as to the value of pneumoperitoneum in the treatment of severe obstructive emphysema.

Pressure breathing has entered the therapeutic field during the past five or six years. Such treatment requires a special apparatus, the "Bennett Intermittent Positive Pressure Breathing Unit", and this machine has been widely acclaimed. Originally it seems to have proved its worth in the prevention and treatment of industrial pulmonary disorders. It is reported that the machine will insure a more uniform distribution of alveolar air (13), and that it will cause "a form of deep breathing exercise, and physical therapy which provides stretching of the respiratory muscles, and appears to enhance elastic tone and circulation of the lungs, and further provides an aid to re-education of better breathing habits" (14). So far, due to lack of experience with its use, we are not qualified to discuss the clinical value of this apparatus in the treatment of patients with severe emphysema. The machine is expensive but its devotees seem to remain most faithful to it. We have only once seen it in operation—in the case of an emergency, in an effort to rescue an elderly woman who was seemingly dying from respiratory acidosis. The hoped-for elimination of the excessive CO_2 (15) did not occur.

c. *Oxygen administration.*

The therapeutic use of oxygen in patients with emphysema has proved to be a very important medicinal adjunct. The administration of this gas in such patients should be reserved for emergencies arising from a severe lack of oxygen. The physiologic efficacy of the repeated and intermittent use of oxygen by patients with emphysema, be it obstructive or nonobstructive, at home or in a hospital, has never impressed us. It is true that elderly emphysematous patients "feel more secure" when the oxygen tank is available; the benefit derived is usually psychogenic, not physiologic.

Furthermore, it must be remembered that the free use of oxygen can prove deleterious (16). It can induce very high blood CO_2 tensions, associated with mental disturbances leading to lethargy, coma and death. The oxygen may be administered by a nasal tube, by a mask (Barach mask, for instance), in a mixture with helium by a more complex apparatus requiring the attention of an anesthetist, or by the machine devised for pressure breathing.

d) *Antispasmodics and antibiotics*

These medications are indicated in patients with pulmonary emphysema who are prone to bronchospasm (primarily or secondarily), and to recurrent infections of the respiratory tract. Isuprel[1] in 1:200 dilution has proved to be very effective as a bronchial relaxant. The patient must be taught to inhale it properly, either through a hand-worked atomizer or by aerosol. Substitutes, such as penicillin, streptomycin and sulfa drugs are most effective in these patients if given by the aerosol technique (17). If such bacteriocidal agents are to be used, it is important to determine the flora of the sputum before choosing the antibiotic. During the course of treatment, the sputum should be re-examined frequently, since a predominantly Gram-positive sputum may change to Gram-negative one after only three days of penicillin-aerosol therapy. If this happens, the antibiotic must obviously be changed. Antibiotics are often used in our clinic in equal parts with Isuprel.

e) *Expectorants*

These are useful in elderly patients with emphysema who suffer from a chronic cough productive of a very tenacious sputum. Potassium iodide (10–15 drops of a saturated solution, four to six times a day) is a great favorite of many physicians. This drug may not be tolerated by some individuals, or it may cause intoxication. The latter is usually heralded by the appearance of coryza, pharyngitis and laryngitis, and later an exanthem. It must be remembered that potassium iodide is a "lytic" drug, and that it is a fungicide. Free use of the iodides in patients with an undiagnosed fungus infestation of the lungs can therefore prove very dangerous. We have found the detergent Alevaire very effective in patients with a harassing cough, when the sputum is scant. It is best administered by aerosol. This detergent can be used in conjunction with Isuprel (or another antispasmodic) and an antibiotic. The following combination has proved to be very useful in our clinic: Isuprel (1:200 dilution) 1 part; antibiotic (perhaps 50,000 units of Crysticillin in 1 cc. sterile water) 1 part; and Alevaire 2–3 parts. The flow of oxygen is regulated at 8 liters per minute. The patient inhales this mixture every four to six hours while awake.

f) *Bronchoscopy*

This is a procedure which is indicated in every patient in whom respiratory embarrassment (not of cardiac origin) develops rather suddenly. It should also be carried out in all elderly individuals with breathlessness of intrabronchial origin, in order to rule out primary or complicating tumors of the air passages. Furthermore, it has been reported that, when bronchoscoped, almost 50 per cent of patients with asthma and emphysema show some degree of bronchial stenosis (18).

Bronchoscopy is a very valuable therapeutic procedure. By this method it is possible to aspirate the thick, inspissated mucus which cannot be coughed up. During such a procedure Isuprel can be easily instilled into the bronchii. In

[1] Isuprel is α-(isopropylaminomethyl) protocatechuyl alcohol.

our experience repeated bronchoscopy has been a life-saving measure in emphysematous and asthmatic patients who are semicomatose for one reason or another. In such instances the bronchoscopy may have to be repeated every six hours for one or two days, despite frequent high tracheal suction in the intervening hours.

DISCUSSION

The reported studies concern 25 elderly patients with emphysema, in whom it was possible to investigate the degree of pulmonary insufficiency thoroughly. The incidence of pulmonary emphysema in the general population is not known, because reliable statistics are not available. Some clinicians believe that obstructive emphysema will develop in from 50 to 65 per cent of asthmatics; others report a frequency of less than 1 per cent in these patients. Obviously both estimates are extreme. From a study of 6,752 asthmatics in the Duke University Clinic we believe that the incidence of obstructive emphysema in the well managed asthmatic who reaches the age of 45 years, is 35 per cent. The per capita incidence of nonobstructive emphysema is even more difficult to ascertain, as is also its etiology. A fairly recent study reports that chronic bronchitis was present in 49 per cent of the cases (19). This suggests the importance of obstructive factors.

It is also impossible to learn the mortality rate from emphysema at any age. We recently wrote to Dr. Bonnett, the Medical Director of the Metropolitan Life Insurance Company, for information regarding the mortality from emphysema. The Manager of the Division of Insurance Medical Statistics very kindly replied, and his answer is quoted: "There is nothing in life insurance statistics that will permit an answer to your question. According to the current 'Manual of the International Statistical Classification of Diseases, Injuries, and Causes of Death,' deaths from emphysema are charged to the category 'Other Diseases of Lung and Pleural Cavity'. This category includes a number of other causes and no split is available to show the proportion directly attributable to emphysema. Where emphysema appears on the death certificate with either chronic bronchitis or tuberculosis or as an early complication of trauma, the death is charged to the underlying basic cause, and it is impossible to determine its prevalence as a contributory cause of death."

The recognition of emphysema is made more exact if a thorough medical history and physical examination are supplemented by x-ray and fluoroscopic studies of the chest. During fluoroscopy the degree of diaphragmatic movement is often astonishing in these patients whose diaphragms are found to be low on physical examination. This corresponds to the vital capacity of these individuals when it is measured by the spirometer, and they are allowed to exhale for a longer time than required in the standard test.

The pathogenesis of nonobstructive emphysema is still not clear, and the cause of the obstructive type is still widely discussed. The resultant physiologic changes have been clarified to a great extent by the advancement in "pulmonary

function" tests. Yet, even the combined knowledge gained from these investigations is not enough to explain the cause or the import of all the abnormal physical and functional developments. A recent article (20) emphasizes the importance of the damage to the pulmonary vascular bed as the determining factor in the degree of pulmonary insufficiency.

The treatment of elderly patients with emphysema entails a willingness of the doctor to educate those who seek his advice. In many instances this requires the correction of purely psychosomatic complaints. The patients, on their part, must work actively in the hope of rehabilitation. The good effects obtained from the breathing exercises will illustrate their desire to improve.

In some instances "pressure breathing" is an effective therapeutic adjunct. This form of treatment cannot be generally dispensed, and it is not available to most patients at home. Pneumoperitoneum is a procedure which has been beneficial in some patients with the obstructive type of emphysema, or the senile type when the diaphragms are extremely low and the abdominal muscle wall offers no support. This again is a form of treatment which requires repeated visits to a clinic and close medical supervision. The beneficial effects, in our experience, do not warrant the cost or the hardship to the patient.

The use of oxygen and helium in patients with chronic pulmonary emphysema should be reserved for selected individuals. It is undoubtedly a life-saving adjunct when acute or prolonged oxygen deficiency develops. It must be remembered, however, that there are great dangers associated with the extended or indiscriminate repeated use of this gas. The inhalation of a mixture of helium and oxygen is of questionable benefit and requires a rather cumbersome procedure.

Bronchoscopy is not only important in the diagnosis of certain instances of respiratory embarrassment of bronchial origin in elderly people but is also of great value in the treatment of patients with pulmonary emphysema. Often it is a life-saving measure, since it allows the aspiration of inspissated mucus and the introduction of an antispasmodic. In one of our patients (to be reported separately) it was necessary to supplement bronchoscopy by the use of an iron lung. This woman, who was 72 years of age, was unable to breathe because of weakness and finally lapsed into coma in profound respiratory acidosis. After forty-eight hours in the iron lung she revived and is still living.

Expectorants, such as potassium iodide, are helpful but must be used with discretion for obvious reasons. The expectoration of mucus, particularly in elderly people, is facilitated by the use of a detergent. This detergent may be added to an antispasmodic in combination with an antibiotic and administered by the aerosol method.

We have not mentioned the role of the corticotropic or corticosteroid substances in the control of emphysema because they are only useful as therapeutic adjuncts in acute emergencies, arising usually in cases of asthma. In these patients the emphysema, although often severe, is secondary in symptomatic severity to the acute bronchospasm.

SUMMARY

Pulmonary emphysema is a common and usually irreversible ailment. It leads to severe pulmonary insufficiency, which makes "pulmonary cripples" of many elderly people. The effective treatment of this disease depends on a well planned long-range program which requires the intelligent cooperation of the patient.

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THE STATE OF THE GASTRIC MUCOSA OF ELDERLY PERSONS WITHOUT UPPER GASTROINTESTINAL SYMPTOMS

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There is considerable documented evidence to show that the incidence of achlorhydria increases with advancing age (1). Often this has been interpreted as a normal or expected consequence of aging. In other words, it has been assumed that either some morphologic form of glandular atrophy or functional type of parietal cell incompetence is a normal process in the geriatric age-group. We shall not attempt to review the literature at this time, but it may be noted that in one of the most recent discussions of the matter (Holler (2)) it is stated that normal aging of the stomach includes mucosal atrophy and fatty degeneration and thinning of the muscularis propria.

The problem is of fundamental importance in an understanding of the gastritides. Histopathologic evidence necessarily had to await the development of a technic for safe and technically convenient removal of biopsy specimens from the stomachs of healthy older persons. There are important objections to judging the microscopic health of the gastric mucosa from autopsy material, as the premortem events associated with death frequently are accompanied by gastric muscular relaxation and distention of the stomach by enteric reflux. Relaxation of the muscularis mucosae and passive distention may lead to confusing mucosal thinning. More important, both premortem agonal and postmortem processes are notorious for their artefactual influence on mucosal histology. Thus, in spite of the abundance of autopsy material, conclusions drawn from critical microscopic studies thereof have not been considered valid for estimations of normalcy. Because of expressed uncertainties over the effects of clamping and ligation on the histology of the mucosa, resected stomachs, too, appear to be unsuitable or perhaps merely untrustworthy material for critical study. On the other hand, biopsy specimens obtained either at gastroscopy or by the vacuum-tube technic, appear to be largely free of artefact and lend themselves well to investigation of mucosal normalcy (3).

This study was initiated in order to evaluate the histology of the gastric mucosa of elderly people who subjectively believed their upper gastrointestinal tracts to be normal. The basis used for comparison was a previously studied group of younger people, selected and examined by the same criteria and methods (3). This control group numbered 105, and the number of subjects per age-decade from the second through the seventh was, respectively, 18, 42, 30, 8, 5 and 2.

MATERIAL

Vacuum-tube gastric mucosal biopsy specimens from 30 persons more than 60 years of age were examined. The oldest subject was 84 years old, and 25 were in their seventh decade. There were 2 women and 2 Negroes.

TABLE 1

Thickness (mm.) of Mucosal Layers as Measured on Stained Sections: Comparison with Specimens from Younger Control Subjects

	Present Series		Controls	
	Range	Average	Range	Average
Total mucosa	0.76–1.14	0.90	0.60–1.14	0.91
Muscularis mucosae	0.08–0.23	0.10	0.03–0.21	0.07
Gland layer	0.36–0.71	0.60	0.39–0.91	0.61

No patient had complaints referable to the upper gastrointestinal tract. None was a true volunteer, but none had any disease which is suspected of influencing the health of the gastric mucosa. Most had undergone investigation of the upper gastrointestinal tract in a search for either a primary lesion to explain metastatic malignant disease, or for an explanation of melena. In all the subjects, roentgenographic study did not show any intrinsic disease of the stomach. Gastroscopic examination gave normal findings in all but 2, there being moderate atrophic gastritis in these. Gastric analysis following histamine stimulation was performed in 23 instances, and in 4 no free acid was found.

HISTOPATHOLOGIC FINDINGS

The specimens of stomach tissue were obtained from the anterior wall of the pars media, close to the greater curvature, approximately in line with the axis of the esophagus. They were uniform tissue discs, measuring from 3 x 5 mm. to 5 x 7 mm. in diameter, and including full mucosal thickness plus a small amount of submucosa.

Data on the thickness of the mucosal layers, as measured on fixed and stained sections, are presented in Table 1. When compared with specimens from the younger control group, no significant differences were found.

The muscularis mucosae was normal in all specimens, without fragmentation, infiltration or hyperplasia. The table shows a tendency toward increased thickness, but this degree was not considered significant.

Except for 3 specimens, the glandular elements were entirely normal in regard to cellular composition and abundance, intestinal metaplasia, and cystic change. In 3 there was relative glandular atrophy, with simple shortening of the glandular portion of the tubules, but morphologically normal chief and parietal cells were present. Each of these 3 stomachs had secreted more than 20 mEq./L of free acid in response to histamine stimulation.

The foveolae were normal in all sections. Intraluminal cell-casts were present in 4 instances.

The neck region (glandular-foveolar junction) was normal in all cases. There was no necrobiosis.

The surface epithelium showed abnormality in only 2 instances. In these, scattered goblet cells were found. There was no nuclear degeneration or scattering.

The only notable features of the lamina propria within the glandular layer were simple edema in one section and small amounts of scattered fibrosis in another. Otherwise, this portion was normal.

The lamina propria within the foveolar layer was normal in 21 patients. Six sections contained moderate edema between the foveolae, with a mild excess of round-cell infiltrate. Two others contained moderate diffuse round-cell infiltrate, and a third showed scattered, poorly defined aggregations of plasma cells and lymphocytes.

COMMENT

Normalcy was, quite obviously, the important finding. The deviations from the normal or expected picture for younger people were minor. There was no instance of atrophic gastritis. The mucosae of the 4 patients who showed no free acid upon histamine stimulation were entirely normal, a frequently encountered phenomenon of mucosal biopsy investigations.

This fact, plus the appearance of all the specimens considered together, suggests that an increase in the incidence of achlorhydria among older people is due entirely to a physiologic abnormality. The nature of the failure is not clear but, wherever the breakdown occurs, it is not reflected in any simple morphologic change in the parietal cells. It is important to note that achlorhydria is in many persons an ephemeral phenomenon, and that serial gastric analyses with optimum stimulation may reveal achlorhydria on some occasions and normal free-acid production on other occasions—further indication of the physiologic vagaries of gastric secretion.

SUMMARY AND CONCLUSION

Biopsy specimens were obtained from the gastric mucosae of 30 elderly persons who had no complaints referable to the upper gastrointestinal tract. No significant disease was found upon histopathologic examination, and the diagnosis of gastritis could be made in none. The specimens showed no more deviation from the usual or normal histologic picture than did specimens removed from younger people.

No evidence was found to support the view that mucosal atrophy, atrophic gastritis, or any generalized mucosal disease should be considered a normal consequence of aging.

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ESSENTIALS FOR REHABILITATION IN THE AGED*

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Improvement in medical care over the past half century has been rapid and gratifying in many respects. The conquest of infectious diseases, the preventive measures instituted, the improved nutritional status and the better socio-economic conditions in our country have guaranteed a longer life span for more people. Economic security and survival to a ripe old age are not sufficient, however. Enjoyable living, free from discomfort, plus usefulness, must be the goal in order to make the sunset years of existence a period in life to be anticipated rather than dreaded.

There are now approximately twelve million persons in this country over 65 years of age (1). It is estimated that the number will be doubled by 1980 (2). This aging population brings new problems in medical care. The medical profession has been alerted and is tackling these problems with vigor and determination. Senility can be and will be made a more livable period in man's existence.

Intensive study is being directed toward the solution and treatment of chronic disease, the incidence of which parallels the increase in the aging population. The nutritional problems, so prevalent among the aged, are being further investigated. The psychosomatic aspects of geriatrics are better understood. Socio-economic conditions are receiving more attention (3).

Aging is not an uniform process. It has its virtues and it has its drawbacks. Degenerative processes are inevitable, and they are usually complicated by chronic diseases. These diseases are incipient in their onset, usually asymptomatic in the early stages, atypical in their progress and prone to lead to disability as they approach clinical recognition (4).

It is true that the preventive aspects of geriatrics must of necessity begin in early life. The tissue changes resulting from disturbed mechanics, infections, nutritional disorders and either psychic or traumatic damage, will cause some impairment of function. The prevention or early correction of these disturbances will lessen the disabilities encountered in later years. Medical supervision must constantly be on the alert for early recognition of variations from the normal. Prompt correction of these variations at an early stage will prevent disability later in life.

Geriatrics carries on this anticipatory attitude toward disability. It initiates corrective measures to stem further advancement. In this sense, geriatric rehabilitation becomes a part of the preventive program of old age.

Rehabilitation at any age must be carefully planned and executed to achieve its greatest success. This is particularly true in the aged individual, in whom

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certain essentials are basic requirements. Rusk (5) and others have pointed out that much can be accomplished when a well rounded program is carried out.

The first essential is a thorough evaluation of the patient and his disability. Chronic disease rarely presents itself as a simple uncomplicated picture. Multiplicity of diseases is the rule, rather than the exception. This picture is further complicated by aging itself. Therefore, a careful study of the patient is needed.

A detailed history is most essential. The initial story as related by the patient is rarely complete. Memory is elusive, faulty, and at times misleading in an older person. For this reason, a questionnaire type of history (6) is valuable, as well as time saving. It must be complemented by additional data obtained from the patient and his close associates. The thoroughness with which this investigation is made will prove therapeutic to the patient. It will also give personality and environmental information that will be highly valuable in rehabilitation (7).

The same thoroughness must characterize the physical and mental appraisal of the aged person. The physical examination must not only give a correct picture of the underlying pathologic process but must also evaluate his stamina and mental capacities. It is important to know the patient who has the disability.

Aging does not follow a charted course. Variations occur in all tissues and organs. When chronic disease is superimposed upon this variable background it is no wonder that early recognition of some of the disease processes is impossible (8). For this reason, frequent re-evaluation is necessary.

The nutritional status of the aged patient is equally hard to appraise (9). Subclinical deficiencies are very common and will retard a program of rehabilitation. The same is true of obesity, which is a handicap at any age, but especially so in the aged. These nutritional difficulties must be corrected. Feeding supervision is just as important in the aged as it is in early life, and frequently harder to get across to the patient. Dietary advice may be just as badly needed for the aged shut-in as for the growing child. Vitamin and protein deficiencies are common, as emphasized by a recent field survey in California (10).

Excesses in food intake may be both qualitative and quantitative, frequently both. Deficiencies may exist along with caloric excesses. Krusen (11) has stressed the importance of a well balanced diet in weight reduction. Exercise contributes very little, except to keep the individual properly conditioned while the reduction in caloric intake lowers the weight.

Functional conquest of disabilities may likewise be greatly influenced by various environmental factors. The socio-economic status, the educational background, the skills developed, the tolerance for exercise and the attitude of home companions are all important.

Rehabilitation is largely concerned with improving physical activity and usefulness. The individual who has held a desk job most of his life can not be expected to show the same tolerance for exercise that a more active person would exhibit. On the other hand, the desk worker, due to his educational background, may be able to grasp new ideas more quickly and thus make the same rate of progress over a given period of time as the person who has to be shown over and over again how to perform a certain activity. The skilled cabinet worker or the

seamstress can be expected to make faster progress in certain types of occupational therapy than a person with a professional background. Therefore, more individual supervision is required in rehabilitation work in the aged.

The attitude of home companions can also influence the ultimate outcome. The early steps in teaching balance or crutch-walking may be spoiled by unfortunate comment. Too much sympathy or over-cautiousness, or the creation of fear can do much to wreck a program. The encouragement of martyrdom paves the way to invalidism.

The changing pattern of home life has its influence on rehabilitation in the aged (12). Former generations revered and respected older people. They were an integral part of the home. They had a place of usefulness and esteem; their idiosyncrasies were tolerated, their experience and judgment were respected. They were the link between the past and the present, with the grandchildren forming the connection with the future.

The present generation is witnessing the breakdown of the close family ties of previous years. We are living in the atomic age, in which the young scientist becomes the instructor of his elders. New frictions are created both in industry and in the home.

The improved economic status of elderly people, either through frugal saving or through social security, or both, has contributed to the weakening of the family unit. The aged, no longer dependent upon their offspring for the necessities of life, are a generation more or less tied together by common interest and social life. They crave this independence and the privilege of living their own lives. They prefer their own home, be it ever so humble or unhygienic.

The attitude of children toward their parents has likewise changed. Individuality has been stressed from birth to adult life, and independence has been emphasized. A well rounded personality, able to cope with present-day problems has been nurtured. Much of the instruction formerly given in the home is now provided outside the family circle. Modern travel and the change from a rural to an urban society have broken family ties. There has been a dispersal of the family unit. The close contact between parent and child has been broken, and each leads a more independent existence.

This changing socio-economic status of the family unit makes each generation stick more closely together. Thus we find elderly people living alone, or with their marital mates or some other individuals of approximate age. They prepare their own meals, which of necessity must be in small quantities. Their frugal habits will not tolerate waste and frequently dietary deficiencies arise due to poor variety in the food. The tolerance for exercise also decreases with age and all too frequently the elderly group will narrow their environment down to the point where activity is greatly restricted and mental stimulation by social contact becomes limited to their immediate companions.

This type of existence goes well until catastrophe in the form of illness or injury strikes. Then comes the hour of decision. Frequently the desire not to be a burden on others, the fear of invalidism or else the inability to adjust to the presence of disability will lead to a depression that may destroy all attempts at rehabilitation.

A program to overcome disability and to avoid invalidism is greatly influenced by the attitude of the individual concerned. There must be some motivating factor to stimulate the aged person to fight for recovery, and this motivation must be sought for and nurtured. It may be the assurance that through hard work he can again be independent, or at least take care of his daily needs. It may be an appeal to his courage to demonstrate that he can fight and overcome his crippling. It may come through patience, persistence and the personal interest shown by all who try to help him. Motivation must be established; without it, rehabilitation becomes almost impossible.

A close physician-patient relationship is most essential. The technical supervision of rehabilitation can be delegated to others but the confidence and inspiration coming from the personal medical advisor is an essential part of the program. The patient himself must not be forgotten. He will need encouragement, sympathetic understanding and guidance to travel the slow road toward functional recovery.

The rehabilitation program, too, must be well planned and executed (13). All personnel engaged in the program must have a sympathetic understanding of the aged. The patient's tolerance for exercise must be watched and the program modified to avoid exhaustion, with the resulting discouragement. More individual supervision is required than in younger persons. An atmosphere of optimism regarding the ultimate outcome must be maintained. Stimulation, by contact with other individuals with similar disabilities who are making progress, is helpful. On the other hand, contact with discouraged individuals may be very detrimental. All of these factors must be considered in working out a successful program of rehabilitation in the aged.

CONCLUSION

Rehabilitation is an integral part of geriatrics, but in treating the disability, the patient must not be forgotten. He needs the human touch and the inspiration of his personal medical advisor. Frequently, the motivation so necessary for success comes from this source. A thorough knowledge of the patient, his nutritional background, his environment, his future aspirations and his capabilities, is the foundation upon which a successful program of rehabilitation can be constructed.

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MAJOR DISEASES OF THE ABDOMINAL AORTA

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Interest in the surgical approach to diseases of the aorta has led to more positive treatment of conditions which have been thought more or less hopeless. Chronic diseases of the abdominal aorta are in the main due to degeneration. Progressive obstruction of the aorta at its bifurcation by arteriosclerosis, and degeneration of the medial coats of the artery with aneurysmal formation are the two results of degenerative disease of the aorta. Inflammatory disease of the abdominal aorta, which is less common, varies in its prevalence as reported by different authors. Kampmeier (1) reported syphilis as the etiologic factor in 57 per cent of aneurysms involving all parts of the aorta. Blakemore (2) in 1947 reported syphilitic etiology in 19 per cent of aneurysms of the abdominal aorta. Linton (3) in 1951 reported no cases of syphilis among 27 such aneurysms. In our experience, arteriosclerosis has been the basis of all aneurysms involving the abdominal aorta.

Aortography has been most valuable in studying diseases of the abdominal aorta, outlining the extent of aneurysmal involvement in relation to the renal arteries and, in addition, demonstrating the segmental nature of chronic obstruction of the bifurcation of the aorta—the basis for surgical consideration of correction of these diseases. Arteriography in cases of acute obstruction has not been widely used in this country, but the French have made wide use of it in the diagnosis and localization of peripheral emboli.

CHRONIC OBSTRUCTION OF THE ABDOMINAL AORTA DUE TO ARTERIOSCLEROSIS

At the outset, it must be realized that an occlusion of a major vessel by arteriosclerosis is usually accompanied by involvement of the arteries in other parts of the body. Moreover, the symptomatology due to chronic obstruction of the bifurcation of the aorta is not entirely dependent upon the degree of occlusion of the vessel, but also upon the effectiveness of the collateral vessels which have formed to short-circuit the obstruction. If the arterial occlusion is a segmental one with the vessels distal to the obstruction being patent, and has developed over a long period of time, the collateral circulation will usually provide an adequate blood flow and the symptoms may be relatively moderate. One of the first symptoms noted in chronic obstruction of the abdominal aorta with good collateral circulation is fatigue of the lower extremities. Bilateral claudication is usually present early, and as long as the segment of obstruction is limited to the bifurcation of the aorta, there are usually no changes in the distal part. In younger males, loss of sustained erectal power is an important symptom of occlusion of the bifurcation of the aorta. The syndrome resulting from segmental occlusion of the aortic bifurcation was first realized as an entity by Leriche (4) in 1940.

Back pain or back claudication is seen in a certain percentage of the cases and may be one of the presenting complaints. Pathologically, the terminal aorta or the two common iliac arteries are involved in the degenerative process, ulceration of the intima and atheromatous plaques producing subsequent progressive thrombosis leading to occlusion. As the thrombotic process advances the symptomatology becomes more severe, because additional collateral vessels are progressively involved.

The relation of Buerger's disease in younger patients with a history of inflammatory arteritis is not clear. Certainly Buerger's disease and arteriosclerosis may exist in the same patient. However, more extensive study of younger patients by means of arteriography and surgical exploration has revealed that many of the patients who were considered to have Buerger's disease in reality have arteriosclerosis.

The diagnosis of chronic obstruction of the terminal aorta is made on the basis of the physical as well as the x-ray examination. Physical examination which reveals the loss of both femoral pulses in a patient who shows no evidence of ischemic changes makes the diagnosis of such a segmental block. If advanced ischemic changes are present, the disease is diffuse and the obstruction is not limited to the bifurcation of the aorta. Aortography by the translumbar route provides accurate information regarding the extent of the obstruction, as well as the size of the collaterals and the size and state of the vessel distal to the obstruction. Considerable attention to detail is necessary in interpreting such x-ray studies, as several factors may cause errors. The injection of an inadequate amount of dye, or poor timing between the injection and making the x-ray exposure, may give a false impression as to the state of the collaterals and the degree of filling of the distal vessel. In some cases, retrograde arteriography through the femoral arteries is necessary in order to determine accurately the state of the vessels distal to an obstruction at the bifurcation of the aorta.

The medical treatment of chronic occlusive disease of the abdominal aorta has been varied and empirical. In the main, it has consisted of the so-called dietary approach to arteriosclerosis (which is controversial and seemingly without benefit) plus the use of vasodilators. Sympathectomy has its best results in those patients who have good development of collateral circulation. This method of treatment is reliable largely in providing protection of the skin from ulcer formation. The relief of claudication has been disappointing in cases with obstruction of the terminal aorta, as many of these patients have thigh claudication as well. Surgical approaches to clean out the obstructed segment have been pioneered by dos Santos (5) and others. Wiley (6), in this country, has reported his experience with thrombo-endarterectomy of these major vessels. Certainly thrombo-endarterectomy is most likely to succeed in a larger vessel such as the aorta or the common iliacs, in which the resultant lumen is more apt to remain patent, because of the large diameter and the velocity of blood flow in this area. The long-term results of this procedure have yet to be defined. Some of the difficulties encountered following thrombo-endarterectomy are due to the weakened vessel wall. To combat this, Wiley has used fascia lata wrappings to rein-

force the wall. The determining factor in any of these direct surgical approaches to occlusion of the bifurcation of the aorta is the extent of the disease both proximally and distally. Therefore, the application of thrombo-endarterectomy is somewhat limited. Resection of the involved segment (arterectomy), as proposed by Leriche (4), has been used; it has usually been combined with sympathectomy. Elkin (7), Ortner (8) and Sautot (9) have reported on these various methods of treatment for chronic obstruction of the aortic bifurcation.

The maximum is accomplished with resection of the segment and its replacement with an homologous artery graft, thus restoring the continuity of the flow. This operation apparently first was performed by Oudot (10, 11) in 1951. In March of 1953 Oudot and Beaconsfield (12) again reported on their experience with resection of the bifurcation of the aorta in 5 cases. This operation has also been performed by us in 5 cases. In 1 case restoration of flow was obtained with the return of pulses bilaterally and complete relief of claudication. In another case the pulses were returned in one extremity, the other extremity being the site of a more distal segmental occlusion in the external iliac artery, not diagnosed previous to resection. In a third case, pulses were similarly restored to one leg with relief of claudication on that side, the other extremity remaining unchanged. In the remaining 2 cases pulses were restored bilaterally at the ankle.

The limiting factors in resection of the terminal aorta with replacement by vessel graft have not been alluded to before. These are 1) the condition of the artery above and below the obstruction and 2) the presence of an additional obstruction distally, which reduces the flow of blood and favors thrombosis of the graft or remaining artery on that side. However, proper selection of cases by means of careful aortography, plus clinical selection of those patients having neither ischemic changes nor rest pain, should result in a number of successful results by this method. The ultimate fate of homologous artery grafts used in cases of this type has not been determined. Moreover, there is no assurance that the artery above and below the graft may not be the site of future disease and obstruction. However, the placement of such a graft does relieve the symptoms and, if successful, prevents the propagation of the thrombus in the obstructed segment.

ANEURYSM OF THE ABDOMINAL AORTA

The etiologic factor in aneurysms of the abdominal aorta has been arteriosclerosis in practically every case. Syphilis does not account for many of these aneurysms, except in certain areas. The exact incidence of abdominal aortic aneurysms is difficult to estimate. From the collected cases in the literature it is certainly very small. However, these represent hospital cases in the main and the impression is that there is an increasing incidence. The importance of this condition, even though the incidence is small, lies in the fact that it is practically always eventually fatal. In the collected series of Estes (13), 63.3 per cent of the patients died from rupture of the aneurysm. Pathologically, aneurysms of the bifurcation of the aorta represent an arteriosclerosis, the end result of atheromatous plaques with ulceration of the lining of the aorta and subsequent de-

struction of the intima and media with expansion of the adventitial wall resulting in aneurysmal formation. Most arteriosclerotic aneurysms of the abdominal aorta are fusiform in nature, usually arising just below the renal arteries (which they may involve). The iliac arteries usually emerge from the aneurysmal sac.

It is important to realize that the only symptom referable to abdominal aneurysm may be the recognition by the patient himself of some change in belt size. It has been stated that arteriosclerotic aneurysms rarely cause erosion and subsequent back pain. However, this was a prominent symptom in 2 of our patients who were subsequently operated upon. Vague symptoms, such as referred pain to the leg and various abdominal complaints, are the usual picture. Finding a pulsatile mass in the abdomen verifies the diagnosis. The clinical progression of aneurysm of the abdominal aorta varies. There are a number of cases on record in which the patient has been followed for years without any particular change in the size of the aneurysm. The majority, however, increase in size. The onset of severe pain, either in the back or referred to the thighs or groins, is a signal of imminent rupture or partial leak. Pulses are usually present distal to the aneurysm. The patient rarely complains of claudication in any form.

The symptomatology changes abruptly when a complication such as massive thrombosis, rupture or dissection occurs. If the aneurysm gives rise to dissection

FIG. 1. Scout film of abdomen showing calcific deposits outlining the wall of the aortic aneurysm.

of the aortic layers, the course becomes acute and severe, and progresses rapidly. A shock-like picture with progressive occlusion of one or both iliac arteries may be seen; with dissection upward, there is pain in the chest.

A dissecting aneurysm may simulate an embolus, either in a common iliac artery or the aortic bifurcation. In differentiation, x-ray examination may be of aid by showing calcification of the aneurysmal wall or the displacement of viscera —the stomach or duodenum in the upper abdomen, the ureters or small bowel in the lower abdomen.

Although the diagnosis of abdominal aortic aneurysm can be made in almost every instance on the basis of the physical examination and ordinary x-ray examination of the abdomen (Fig. 1), aortography is now frequently used to study the extent and nature of the aneurysm and to help determine whether operative treatment is possible. Aortography may be performed by translumbar puncture (Fig. 2), or through a catheter introduced from below by way of a femoral artery (Fig. 3). Neither method is without danger, as a portion of mural thrombus may be dislodged or an atheromatous plaque may be pushed off by either the needle or the catheter. Aneurysms of the upper abdominal aorta or lower thoracic aorta have been visualized by passing a catheter, under fluoroscopic control, through the left brachial artery down into the aorta to deliver the radiopaque material.

In regard to physical examination, it should be remembered that an aneurysmal mass may be displaced either to the right or the left of the midline. Aneurysms of the upper abdominal aorta are more difficult to diagnose by physi-

FIG. 2. Translumbar aortogram of aneurysm shown in Figure 1. Thrombosis has occurred, leaving well formed channel through the center of the aneurysm.

FIG. 3. Aortogram by the retrograde method via a branch of the femoral artery, outlining the aortic aneurysm.

cal examination because of the difficulty in differentiating pulsation transmitted by an upper abdominal tumor from the expansive pulsation of an aneurysm. In the asthenic individual with a tortuous abdominal aorta, a mistaken diagnosis of abdominal aneurysm may be made.

Treatment

The nonsurgical management of abdominal aneurysms is obviously limited in the main to restricting the activity of the patient. Occasionally the pain may be relieved by changes in position, such as from the recumbent to the sitting position. Management of associated hypertension should be attempted.

The surgical attack on aneurysms constitutes one of the earliest chapters in surgical history. The use of the proximal ligature, as well as the operation of aneurysmorrhaphy dates back to earliest times. Ligation of the abdominal aorta proximal to the aneurysm, has met with failure, as the constant pressure against the complete ligation of the aorta has almost uniformly resulted in rupture and death. Subsequently, attempts to make the ligation partial were successful to some degree in reducing the flow into the aneurysm.

Spontaneous thrombosis of the abdominal aortic aneurysm apparently rarely occurs, but when it does, it usually results in ischemia distally. We have observed 2 cases in which the preoperative diagnosis was saddle embolism; exploration revealed a small bifurcation aneurysm which had thrombosed. These patients showed complete ischemia of the distal lower extremities.

Several methods have been developed, having as their purpose the stimulation of fibrosis around the aneurysm, thereby decreasing the possibility of rupture, and diminishing gradually the inflow into the aneurysm. Poppy and Oliviera (14) in 1946 reported on their experience with wrapping thoracic aneurysms in cellophane and pointed out that differences in results depended upon the type of cellophane used. Lowenberg (15) used "cutis" grafts to wrap the aneurysm, in order to produce fibrosis and reinforce its wall. In 1 case reported by de Takats (16), in which such a graft was used, it had completely disappeared at autopsy six months later. Berman (17) used 0.9 per cent diacetyl phosphate in olive oil to produce fibrosis. Sheets of polyvinyl sponge to reinforce and stimulate fibrous tissue formation over the aneurysmal sac were used by Grindlay and Waugh (18). As pointed out by Linton and others, the inherent disadvantage of all these methods for the production of fibrosis is that it is practically impossible to extend the procedure to the back of the aneurysm where it is equally important.

The wiring of aneurysms is an old technic which has been discussed in detail in articles by Linton (3) and Blakemore (19). The two different methods of wiring have been extensively used. The first is the "pack" method in which the sac is filled with wire and the presence of a mesh is depended upon to produce partial thrombosis and reduction in activity of the aneurysm. Linton (3) has reported 17 patients who survived this form of wiring. He states that in his most recent cases he has endeavored to utilize the so-called "pack" method to the extent that a "maximum footage of wire was inserted." The second method of wiring utilizes an electric current to heat the wire after introduction, in order to produce thrombosis. This technique has been reported by Blakemore (19) and his experience with it is the largest in this country. He believes that the use of small lengths of wire together with controlled coagulation by electrical means, plus proximal banding of the aorta, is the best approach.

Wiring of the aneurysm probably has the widest application. There is some danger in the "pack" method from wire progressing up the proximal or distal vessels when it is inserted (Fig. 4). In either method there is still danger of leakage and rupture of the aneurysm, although to a lesser degree than without treatment.

The final methods to be discussed are those having to do with restoration of the continuity of the aorta following resection of the aneurysmal mass. Restoration of the continuity without use of a graft is applicable in saccular aneurysms; simple removal of the sac is followed by lateral arteriorrhaphy to restore the continuity and caliber of the vessel (Bahnson (20)). Another method is that reported recently by Freeman (21), in which he used an homologous inlay vein graft placed through the opened aneurysmal sac, and sutured the vein to the proximal and distal segments of the aorta. He then, after re-suturing the aneurysm, introduced blood between the vein graft and the aneurysmal sac, so that it would clot there and add support to the vein graft.

Actual resection of the aneurysmal sac with replacement by vessel graft was first accomplished by Dubost (22) and by Shafer (23), using homologous arterial grafts. In addition, Brock (24), DeBakey and Cooley (25), and the authors (26) have reported on their experiences with the use of homologous arterial grafts

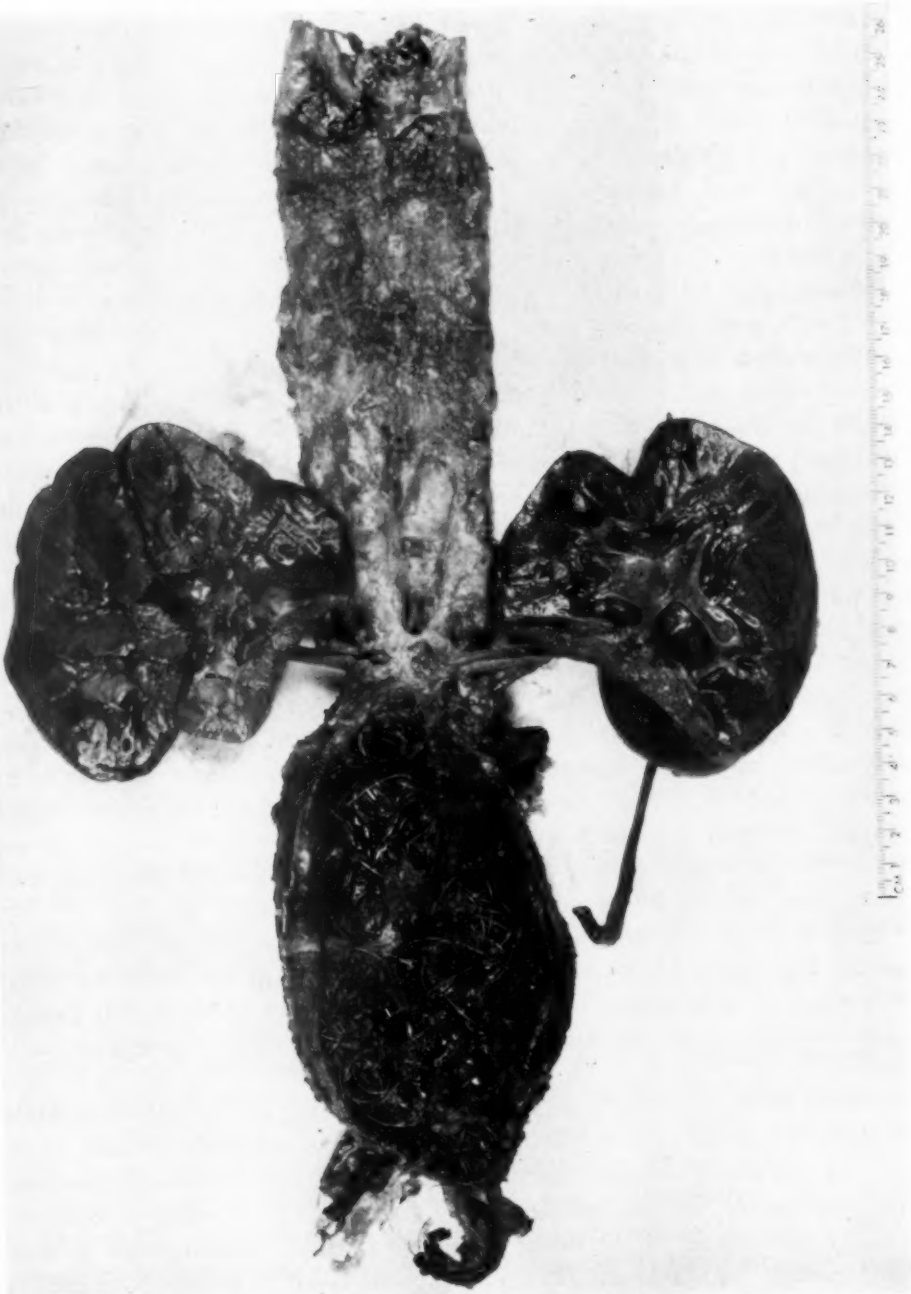

FIG. 4. Autopsy specimen of abdominal aortic aneurysm which had been treated by wiring ("pack" method). Death occurred on the second postoperative day. Photograph shows how wire had been pushed into iliac arteries.

for replacement of the bifurcation of the aorta following resection of an aneurysm. Replacement of the aortic bifurcation almost of necessity requires the use of artery grafts, since a vein graft, unless supported by one means or another, will soon dilate. Experience in this method is limited. We have applied it in 5

cases. The replacement of an abdominal aortic aneurysm by vessel graft is dependent upon several factors, at least at this stage of our experience. First, we have not attempted resection of an aneurysm which involved the renal arteries, although this is not out of the realm of possibility. The aneurysms resected involved the bifurcation of the aorta and extended up to within 1 or 1½ inches of the renal arteries; the iliac arteries emerged from the aneurysmal sac. A bifurcation homologous artery graft was used, obtained from a blood vessel bank stored at minus 70°C. In 3 patients complete circulation was restored to both extremities and they have been followed for from one to six months. In the first case operated upon, dislodgment of a plaque above the proximal anastomosis resulted in partial occlusion of the right external iliac artery. The functional result is one of normal circulation in the left lower extremity; the right lower extremity is pulseless but not ischemic, and the patient suffers only from mild claudication. Subsequent exploration of the obstructed iliac artery revealed old arteriosclerotic involvement as well as the superimposed embolism. Attempts at restoration of the lumen were unsuccessful. This patient has been followed for fourteen months. One death occurred six days following resection for an aneurysm. The graft functioned well following surgery, with return of femoral pulses. However, there was apparent thrombosis of vessels distally in the feet, with marked insufficiency in the lower extremities following surgery; in addition a right hemiparesis occurred during the operation. This patient had severe postoperative atelectasis, necessitating a tracheotomy, with death on the sixth postoperative day.

The proportion of aneurysms in which the resection-graft method of treatment can be applied has not been determined. When it is possible to resect the aneurysm and restore the continuity of the vessel with a homologous artery graft, this would seem to be the optimum treatment.

The limitations in the use of resection of abdominal aortic aneurysms and replacement with artery grafts are dependent upon several factors. The oldest patient we operated on was 63. In order to carry out this procedure the patient must have a good cardiac reserve, since the operation is of fairly long duration. Another limiting factor is evidence of arterial insufficiency of the lower extremity. Reasonably good kidney function is also a prerequisite for the application of this method.

ACUTE OCCLUSIONS OF THE ABDOMINAL AORTA

Primary thrombosis in an otherwise normal aorta is extremely rare. It has been reported in severe cases of diseases such as typhoid fever or amebic dysentery.

Embolic occlusion of the bifurcation of the abdominal aorta constitutes one of the most dramatic incidents in medicine. The most common predisposing factor is intracardiac thrombus associated with either auricular fibrillation or myocardial infarction. The most classic cases are those occurring in auricular fibrillation due to rheumatic heart disease. However, a number of such emboli originate from thrombi which form on atheromatous plaques in the aorta above the bifurcation.

FIG. 5. Autopsy specimen, showing aortic bifurcation embolus in relati vely normal aorta.

An embolus lodging in the bifurcation of the aorta produces varying symptomatology, depending upon whether it is superimposed on a previous partial obstruction due to arteriosclerosis or whether it occurs in a normal tissue (Fig. 5). With sudden occlusion in a previously completely patent aorta, the symptomatology is fairly constant—loss of sensation, coldness and subsequent paralysis of the lower extremities. Subsequently the foot becomes cadaverous and a line of temperature demarcation occurs somewhere in the region between the knee and the mid-thigh, depending upon the collateral circulation. In some cases the line of demarcation may progress into the upper thigh (Fig. 6). Complete loss of pulses in the lower extremities and loss of oscillometric breathing readings confirm the diagnosis. The clinical course in the absence of surgical treatment in a case such as that just described, is that of eventual loss of the limb and probable death.

In patients with previous partial occlusion of the aorta and superimposed embolism one may see a varying picture. There may be viable but cold extremities with retained sensation, or a line of demarcation at one of various levels from the foot up, depending upon the collateral circulation previously formed. In some instances there is a line of demarcation in one leg, but the other leg is

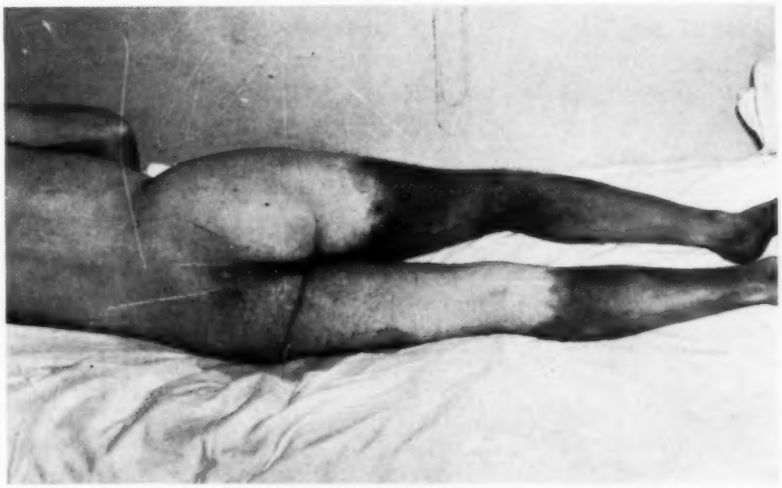

FIG. 6. Line of demarcation occurring following aortic bifurcation embolism

entirely viable. In the case of a dissecting aneurysm at the bifurcation of the aorta, the differential diagnosis is sometimes difficult to make. The patient usually presents more of a shock-like picture and the findings may change from hour to hour as the dissection progresses. One differential point in many cases is that the level of paralysis and loss of sensation may be much higher than the level of temperature demarcation, indicating dissociation between the level of arterial obstruction and the involvement of blood supply to the spinal cord.

In a patient with aortic bifurcation embolism and completely nonviable extremities the only life-saving procedure is that of embolectomy. The reason for the limited number of successful aortic bifurcation embolectomies is the fact that many of these patients are in very poor condition and tolerate any anesthetic very poorly. If the case is that of a classic aortic bifurcation embolism, then the procedure is the direct approach to the bifurcation through a laparotomy incision. We have used spinal anesthesia for this purpose. In cases in which there is a doubt as to whether the obstruction is due to thrombosis superimposed on a previously arteriosclerotic bifurcation (Fig. 7), or whether it is due to fresh embolism, we often explore the femoral arteries first under local anesthesia, in order to determine the state of the arteries in this region. If diseased and pipestem arteries are found, this procedure precludes the necessity of giving a spinal or general anesthesia and exploring the abdominal aorta. An additional danger in patients who have had an embolus superimposed on a partial occlusion is the development of proximal thrombosis of the aorta with ultimate closure of the renal arteries.

We have not used aortography to visualize acute occlusion of the aorta, although this may be of great value in certain cases.

Continuous circulation following aortic bifurcation embolectomy depends on maintenance of cardiac compensation, the use of anticoagulants and the avoidance of hypotensive periods either during or after surgery.

FIG. 7. Thrombosis of aortic bifurcation superimposed on severe arteriosclerosis of aorta (previous incomplete occlusion).

SUMMARY

1. The various major diseases of the abdominal aorta have been discussed and the treatment outlined.

2. Aneurysm of the abdominal aorta is a fatal disease and a radical approach to treatment is justified. Wiring of the aneurysm has resulted in lowering the mortality and there has been more experience with this method than with any other. Resection of the abdominal aorta with replacement by homologous artery graft, when feasible, would seem to be the ideal method of treatment.

3. Chronic occlusion of the abdominal aorta due to arteriosclerosis has been recognized with increasing frequency as an entity. Treatment is far from satisfactory, as the occlusion represents a part of a generalized disease. When the disease is segmental, resection of the aortic bifurcation, with replacement by homologous artery graft, offers hope in selected cases.

4. Embolism of the aortic bifurcation is a surgical problem but must be differentiated from thrombosis superimposed on chronic arteriosclerotic occlusion.

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SENILE MACULAR DEGENERATION: A COMMON CAUSE OF DEFECTIVE SIGHT IN THE ELDERLY

ARTHUR J. BEDELL, M.D.*

Albany, New York

One of the most annoying, depressing and altogether discouraging conditions involving the sight of the aging is a degeneration in the center of the retina and choroid which goes under the name of senile macular degeneration. This condition had been known for many years, but is now assuming a more important position because of the ever-increasing number of the aged.

An affected person first notices that reading, writing and other near use of the eyes is more difficult and that a change of glasses does not help. Often one eye is involved weeks or even months before the other, and unless the patient is a keen observer the disease may be far advanced by the time he seeks help because a similar change has started in the second eye. This difference in time is an important clinical fact which, when elicited, should suggest the possibility of this disease.

The most significant symptom is a dark spot before the eye or eyes, which increases in size. This does not necessarily mean a steady progression, for stationary periods are not uncommon. There are no other symptoms; no redness of the eye ball, no lacrimation, no discharge, or sense of illness. Usually the disease has been progressing for a considerable time, often years, before the patient comes under observation of an ophthalmologist.

There are two distinct types of this destructive, usually binocular affliction, the exudative (Figs. 1, 2, 3 and 4) and the sclerosing (Figs. 5, 6, 7 and 8).

The initial sign in the first group is a hemorrhage in the macular region, which increases in size until it becomes a more or less flat, red clot beneath the retinal vessels. Portions of the primary hemorrhage may be absorbed but new extravasations appear in an ever-expanding ring, until eventually the macular region is encircled by them.

In this exudative type the organized thick scar of connective tissue increases in amount and may at times become so elevated, pigmented, and uneven that it is several times the disc diameter and may lead the unskilled to the diagnosis of a malignant growth.

The true sclerosing type frequently starts with a small, soft, grey macular edema followed by flat granular hemorrhages near the periphery. As the oozing continues the retina and the choroid show changes in pigmentation—decreases in some parts and collections in others—so that the surface is mottled. This is followed by partial disappearance of the pigment layer with the development of a more or less round or ovoid, pale pink area in which the choroidal vessels, smaller than normal, irregular in caliber and often white walled, may be traced. As the disease progresses the atrophic spots increase in number and size or they

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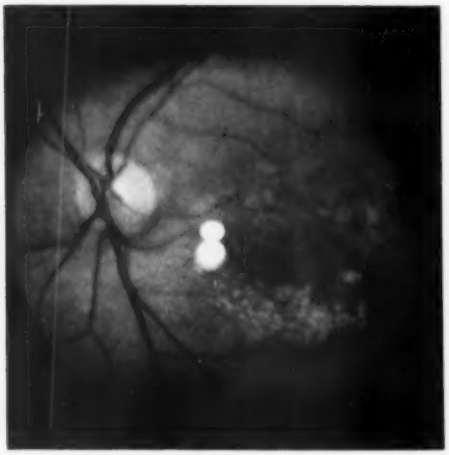

FIG. 1

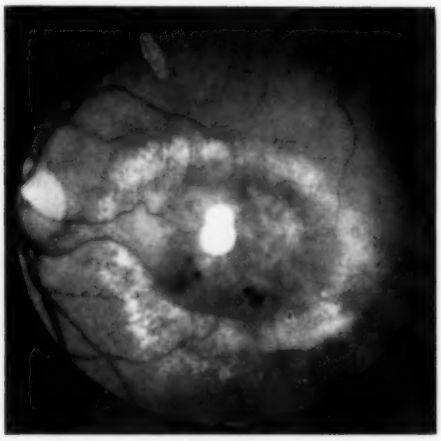

FIG. 2

FIG. 1. *Senile macular degeneration, exudative type.* The fundus of a 58-year-old woman. Left eye vision: 20/100. Early changes in the macula consisting of superficial exudates and granular hemorrhages. (From Bedell, A. J.: The fundus of the eye, *Ciba Clin. Symposia* **4:** 135 (July) 1952.)

FIG. 2. *Senile macular degeneration, exudative type.* Fundus of the patient in Figure 1, four months later. Considerable increase in the retino-choroidal swelling in a crown of denser, less lace-like exudate. (From Bedell, A. J.: The fundus of the eye, *Ciba Clin. Symposia* **4:** 135 (July) 1952.)

may coalesce into a single, large, oval or round, depressed macular defect. The loss of vision is the same in both forms, but in the vascular type the retinal and choroidal vessels show more alterations in width and often also in course.

Whenever a patient of 50 or more years has a prolonged obscuration of vision which interferes especially with close work and is not caused by cataract, glaucoma or any other manifest disease, and cannot be corrected with glasses, senile macular degeneration should be suspected.

If a cloud comes suddenly before the eye of an elderly patient and the diagnosis is made of a hemorrhage in the macular area, a thorough examination with an analysis of the cardiovascular system and an intensive investigation of the ocular fundus must be completed before senile macular degeneration can be excluded, for this is one of the commonest methods of onset.

Once the diagnosis of senile macular degeneration has been made and any cardiovascular-renal changes corrected as far as possible, the patient should be taught how to live and how to evaluate and appreciate the sight he has left. Above all, he should be constantly reassured that blindness will not result from this disease. Macular degeneration with its distinctive, pathognomonic destruction is a prime geriatric problem. The rapidity of change is not the same in all patients, nor is the elapsed time before the second eye begins to fail. The vision in the first eye may be markedly impaired before the patient recognizes any sight loss, even when the defect may have been preceded by a circumscribed transitory cloud, the loss of letters in a word, unevenness of lines, or even a blind spot (scotoma).

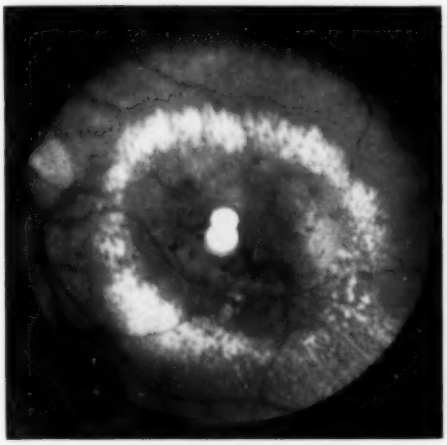

FIG. 3

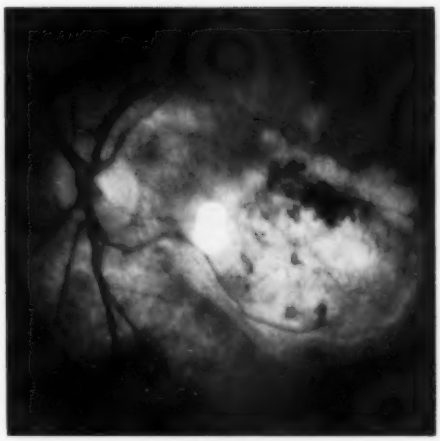

FIG. 4

FIG. 3. *Senile macular degeneration, exudative type.* Fundus of the patient in Figure 1, seven months later. Exudate arranged as a ring enclosing the macula with fresh hemorrhages. (From Bedell, A. J.: The fundus of the eye, *Ciba Clin. Symposia* **4:** 135 (July) 1952.)

FIG. 4. *Senile macular degeneration, exudative type.* Fundus of the patient in Figure 1, four years after onset. The end result—a large elevated, irregular mass with diffuse pigmentation and uneven border. (From Bedell, A. J.: The fundus of the eye, *Ciba Clin. Symposia* **4:** 135 (July) 1952.)

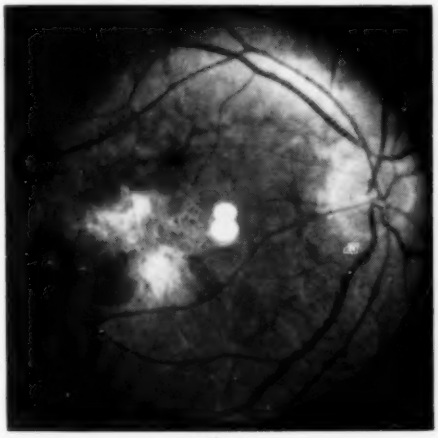

FIG. 5

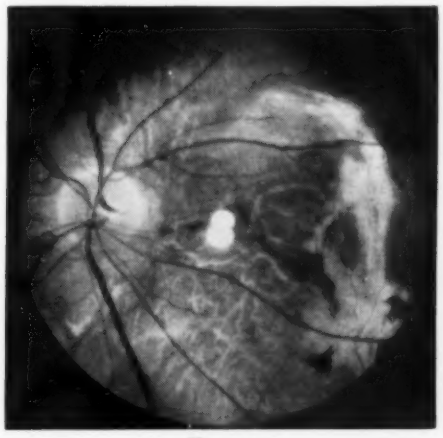

FIG. 6

FIG. 5. *Senile macular degeneration, sclerosing type.* Fundus of a 52-year-old man. Right eye vision: 1/200, with a central scotoma. A flat organized scar with choroidal vessel sclerosis and angioid streaks.

FIG. 6. *Senile macular degeneration, sclerosing type.* Fundus of the patient in Figure 5. Left eye vision, 1/200; marked choroidal vessel sclerosis and a large white scar.

A 73-year-old man came in because of poor vision, saying that he had noticed that his sight had been failing for ten years. The vision of the right eye was 5/200. There was a very extensive macular degeneration with a yellow-grey elevated collar, called retinitis circinata; this is only a phase in development, not a disease

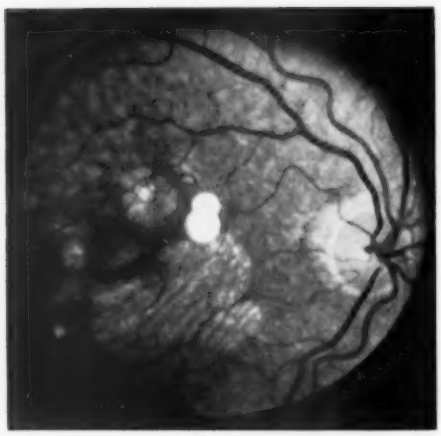

FIG. 7

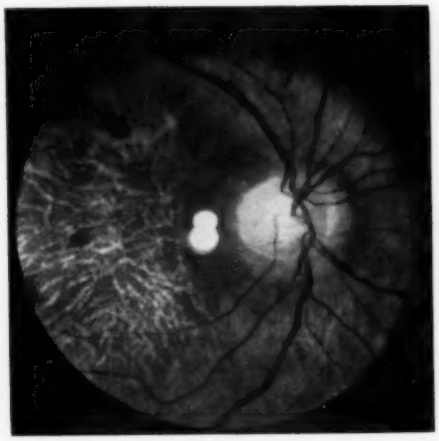

FIG. 8

FIG. 7. *Senile macular degeneration, choroidal vessel type.* Fundus of a 70-year-old man. Many drüsen. Several oval patches from which the pigment has been absorbed, with white choroidal vessels in the background.

FIG. 8. *Choroidal vessel sclerosis.* Fundus of a 65-year-old man. An excellent example of choroidal vessel sclerosis. A large area including the macula shows the extensive vessel alterations. (From Bedell, A. J.: The fundus of the eye, *Ciba Clin. Symposia* **4:** 135 (July) 1952.)

entity. In his left eye the vision was 1/200 and the changes were greater. Eventually there was an immense grey elevation, *i.e.*, exudative senile macular disease.

When a 78-year-old man discovered that he could not see to read, he was found to have a large white mound of organized exudate, on the top of which was a minute granular red hemorrhage.

The sclerosing form assumes many shapes, but the exudative tends to be a round or ovoid mass.

We have several photographic histories showing the progressive changes over a ten-year period, starting as a small macular hemorrhage and terminating in a bulky scar. We also have photographic records of the sclerosing destructions, ranging from macular edema to the depigmented patches.

Early senile macular degeneration of the exudative type must be distinguished from thrombosis of a branch of the central retinal vein; a retinal or choroidal hemorrhage; the retino-choroiditis of tuberculosis, syphilis, sarcoidosis, undulant fever, toxin effects, or other similar cause; and some of the unusual extravasations following trauma. Most of these confusing conditions have specific signs, and difficulties in diagnosis arise only when the history is defective or the examination superficial.

If senile macular degeneration has been present for years, it often looks like a malignant melanoma. To distinguish one from the other occasionally calls for the highest degree of professional skill and an extensive clinical experience.

When an old person has a central scotoma and is able to see but cannot read, senile macular degeneration is possible, for it is much more common than is recognized by physicians or laymen and should be thought of in all cases.

A male 52 years of age showed a central vision of 1/200 and there was a large sclerosed area in each macular region. He had no hypertension, or kidney dysfunction. His visual disorder was simply the result of the death of that particular portion of his eye. It is therefore necessary to recall that tissue age may be entirely different from chronologic age.

Central degenerations in patients with high myopia are by no means rare, but the combination of myopia with senile macular degeneration is quite different. This is easily recognized by the continuing changes which take place—from the initial grey, more or less round, area to the wide circle of depigmentation.

When the sclerosis is marked, with vision reduced to 2/200 in each eye, the choroidal vessels are white and sharply outlined, and the bleached spots are easily recognized.

After the sight is once reduced, it is never restored, for there is no treatment that will cure the disease. Some drugs seem to retard the destructive process. It has been well known for years that small doses of potassium iodide frequently act as a general tonic in the true sense of an uplift. Thus, for decades, 3 grains of potassium iodide three times a day has been the medication of choice. Obviously the general body health must be maintained at the highest possible level.

In all chronic diseases there are periods of remission and this one is no exception. The improvement which follows the administration of iodine, therefore, may be coincident with a recession, or it may be real. Whichever it is, the patient is grateful that something is being done without a great expenditure of money.

It is most important that the physician have a comprehensive understanding of this malady of senescence, so that he can tell the patient "You will never go blind from this disease, although your reading sight cannot be restored." If we place ourselves in the position of the patient who has been noting the steady decrease in reading ability, and think of the mental relief derived from such a statement, physicians should be glad to know more about senile macular degeneration and the prognostic significance of the changes.

The care of the elderly sick is an increasingly demanding situation, for more and more people live longer and enter the period of physical and mental dependency. Anything we can say or do to reduce the burden on those who have to care for them is of advantage, and will necessarily give great relief.

The clinical course of senile macular degeneration must be thoroughly understood and it is our considered advice that no patient should have an eye removed because of a macular mass until photographs have helped to exclude the benign degenerative changes. By comparing fundus pictures taken at two-week intervals, a malignant growth can often be excluded and the eye retained. Eyes are removed every year because the basic nature of senile macular degeneration is not appreciated.

BILATERAL HIP INJURY IN A GERIATRIC PATIENT: A CASE REPORT

BERNARD J. FICARRA, M.D.

The Department of Surgery, Roslyn Park Hospital, Roslyn Heights, Long Island, N. Y.

It has been erroneously considered in some quarters that elderly patients with hip fractures are perpetual cripples. I should like to report a case in which a woman over 80 years of age sustained a bilateral hip fracture and is now ambulatory.

CASE REPORT

Mrs. C. G., an 85-year-old housewife, sustained a fracture of the neck of the left femur in December 1950. The injury occurred when she slipped on a sidewalk. She was taken to the hospital and after preliminary studies and treatment she was operated upon. A Smith-Petersen nail was inserted into the left femoral neck.

FIG. 1. Photograph of X-ray film showing both nails in situ.

One year after this injury (December 1951) she was able to get along without crutches. She was not advised to do so, but she did it of her own volition. Three months after she abandoned the use of crutches, she slipped on her kitchen floor. She sustained an intertrochanteric fracture of the right hip without injury to the other side. She was again taken to the hospital and a Smith-Petersen nail with a Blount attachment was employed to immobilize the fracture sites (Fig. 1). She had an uneventful recovery. Both operations were performed under spinal anesthesia. The patient is now ambulatory, using no crutches and bearing full weight on both legs. She is well at the time of preparing this report (July, 1953)*

*February 1954—the patient continues well.

SUMMARY

1. A case is presented of an 85-year-old housewife who sustained bilateral hip fractures.

2. The first injury occurred in 1950, the second fifteen months later.

3. Each fracture was treated by means of internal fixation under spinal anesthesia.

4. The patient is now ambulatory, does not use crutches and bears full weight on both legs.

AMERICAN GERIATRICS SOCIETY

THE 1954 ANNUAL MEETING

The 11th Annual Meeting of the American Geriatrics Society will be held at the Hotel Fairmont in San Francisco just preceding the meeting of the American Medical Association. The scientific sessions of the meeting will begin Thursday afternoon, June 17, and continue through Saturday morning, June 19.

Hotel reservations should be made through the San Francisco Convention and Visitors Bureau, 200 Civic Auditorium, San Francisco 2, California. Members should reserve accommodations immediately, stating times of arrival and departure, because the hotels expect to be filled to capacity.

The Annual Business Meeting will be held in the Fairmont Hotel, Thursday morning, June 17, at 9 o'clock. The room for this meeting will be announced later. All Scientific Sessions will be held in the Nob Hill Room of the Fairmont, and the annual dinner is scheduled for the Terrace Room on Friday evening, June 18.

The meeting will be open to all members of the American Geriatrics Society and to physicians and other scientists who are interested in the field of geriatrics. The program will cover many aspects of geriatric medicine, and there will be several panel discussions on such subjects as recent developments in cardiology, methods of determining operability in older patients, and the relation of industry to geriatrics. Outstanding clinicians and investigators will participate.

DR. LAURANCE W. KINSELL, Highland Alameda County Hospital, 2701 Fourteenth Avenue, Oakland 6, California, is in charge of local arrangements for the meeting.

W. O. THOMPSON, M.D.
President

THIRD INTERNATIONAL GERONTOLOGICAL CONGRESS—LONDON

The Third International Gerontological Congress will be held in London, England, *July 19–23, 1954*, under the presidency of Dr. J. H. Sheldon. Arrangements by the British Organizing Committee are well advanced. Dr. R. E. Tunbridge, B.M.A. House, Tavistock Square, London W.C.1, is Chairman of the Committee. An *American Committee* is cooperating with the British Committee, especially in regard to the program. Of this Committee *Dr. W. B. Kountz, 660 S. Kingshighway Blvd., St. Louis 10, Mo., is the Chairman.* The Congress will be both instructive and enjoyable. It is imperative for those planning to attend and to present their work, to send titles of papers, together with abstracts of not more than 250 words, to Dr. Kountz. As in the case of other International Congresses, personal invitations to present papers are not necessary but in the event that these would be helpful in obtaining subsidies for travel, the American Committee will be happy to request the British Organizing Committee to send official invitations.

The Research Committee on Biology and Medicine will meet at the same time as the Third International Gerontological Congress. The Chairman of this Committee is Dr. W. O. Thompson, Chicago, Illinois.

Because the time is short and it is difficult to get transatlantic reservations, except the more expensive ones, blocks of reservations are being held by Convoys Travel Service, 1133 Broadway, New York 10, N. Y. All correspondence concerning *travel information* should be addressed to *Mrs. V. Kelly, 660 S. Kingshighway, St. Louis 10, Mo.*

PROVISIONAL PROGRAM

July 19, A.M. Meeting of the Governing Body.
July 19, P.M. Official opening of Conference, followed by meetings of the Sectional Committees.
July 20, 21 and 22. Scientific Meetings of the Conference.
July 23. Closing Meetings of the Conference.

The meetings and discussions will be divided between three main sections of the Conference:

Section 1. Biology and Pathology.
Section 2. Geriatric Medicine and Psychiatry.
Section 3. Sociology and Psychology.

Discussions and symposia involving combined meetings of two or more sections, and meetings of the Research Group, are to be arranged.

Visits

Visits will be arranged during the meeting in London, to centers in and around London. After the Congress, tours will be arranged to other centers in the United Kingdom.

Opportunities will be given to members to see geriatric units in hospitals,

residential homes, housing accommodations, clubs and special industrial facilities.

Proposed Provincial Tours

Following the main Congress, tours will be arranged to enable members to see something of the work for the elderly throughout the country. Local members have expressed the wish to meet members attending the Congress and to entertain them during the provincial visits.

The following tours are being arranged: 1). London, Oxford, then to the West Country, returning via Southampton and Salisbury to London. 2). London, Nottingham, Sheffield, Leeds, Harrogate, Durham, Sunderland, York, Lincoln and Cambridge, to London.

Social Occasions

July 19. President's Reception in the evening.
July 21. Evening Reception.
July 23. Conference Dinner.

Throughout the Congress, visits to centers of scenic and historic interest in and around London will be arranged. Opportunities will also be provided for members to meet informally.